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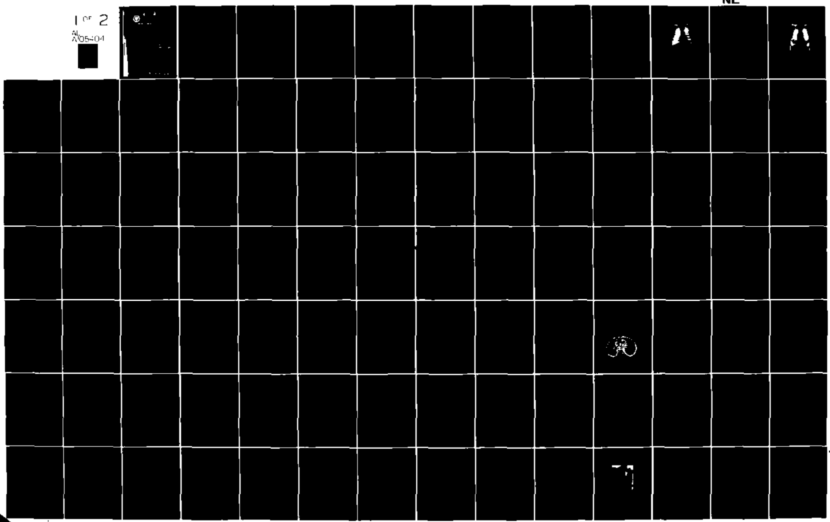

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THE ACQUISITION OF PERCEPTUAL DIAGNOSTIC SKILL IN RADIOLOGY

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Technical Report No. PDS-1

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Five experts, each with at least ten years of on-the-job experience since their residencies, and eighteen radiology residents in the first through fourth years of training diagnosed five standard chest X-ray pictures. Verbal protocols were collected, and an in-depth qualitative analysis of the protocols was performed.

One of the most striking differences between the experts and the residents was in the ability of more experienced subjects to see anatomy;

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20. that is, to build a rich mental representation of the anatomy of the patient whose radiograph is being diagnosed. There were ability-related differences in the extent to which general diagnostic schemas were directly triggered by abnormality features. In many cases, experts detected a general pattern of disease that severely constrained the possible interpretations of otherwise ambiguous film abnormalities.

We concluded that developing a representation of the patient is more of a recursive process in experts. That is, initial film features are recognized as signs of abnormality. They, in turn, drive higher-level reasoning about the likely sources of disease, and that reasoning in turn sets the stage for further feature recognition. This recursion through the different stages of the diagnosis process is most successful and occurs in richer forms in experts. The expert has the ability to sustain the looking and reasoning cycle even in the face of considerable complexity. There appears to be a strong interaction between this recursive ability and their broad general schemas for constraining syndrome information. Another conclusion is that experts are opportunistic planners, in the sense discussed by the Hayes-Roths, with very rich recognition and constructive perceptual abilities. They are very sensitive to new information and know when to seek additional data. The whole course of reasoning in an expert can be changed by the noticing of a single small anatomical detail. Expert subjects were both more able to ignore irrelevant data and more able to take immediate account of relevant information.

The Acquisition of Perceptual Diagnostic Skill in Radiology

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Preface

Because of the extensive nature of this report and the complex domain which was studied, we begin, in Chapter I, with an extended summary of the study being reported and of the conclusions that were reached. It can be read without reference to the remainder of the paper, which presents background, perspective, method, analyses, and theoretical and empirical conclusions in greater detail. Chapter II introduces the problem and reviews relevant literature on radiological diagnosis. Chapter III presents the methodology of the study, and Chapter IV presents a preliminary analysis of the structure of subjects' verbal protocols. Chapter V presents the main results of the study, a detailed, process-oriented analysis of protocols from three different film diagnosis problems. While most of the conclusions of the study are stated in Chapter I, Chapter VI provides a summary of the study and discussion of a few of its implications.

We wish to thank Dr. Bertram Girdany, Chairman, University of Pittsburgh Radiology Department, for his assistance in arranging contact with the residents and with some of the staff radiologists who participated. We also thank Dr. Simon Slasky for many insightful observations that contributed to this work. We especially acknowledge the contributions of Harriet Rubinson, who conducted many of the experimental sessions and did much of the analysis work, and Carlyne Mattoon, who preceded her in that capacity. We also acknowledge the support of the Personnel and Training program of the Office of Naval Research, which funded this work, and the SUMEX-AIM project, funded by the National Institutes of Health at Stanford University, which facilitated many of the contacts that allowed us to learn better ways to pursue this work.

CHAPTER I

OVERVIEW

I.A INTRODUCTION

Diagnosing X-ray pictures, or radiographs, can be very difficult. While some films can be diagnosed by people with a minimum of medical and radiological knowledge, there are many cases with which even experts have great difficulty and a low probability of achieving the correct diagnosis. The general consensus of many radiologists and psychologists who have addressed this issue, is that this is because of detection problems, that some signs of illness are so faint in a radiograph that even experts miss them. In our work, we have been comparing the diagnostic capability of expert radiologists with that of radiology residents. In contrast to previous efforts, we have taken radiology to be largely a problem solving activity and not just a simple detection activity, and have found a variety of differences between experts and novices that go beyond the matter of detection.

The conclusions we report are based upon a study in which five experts, each with at least ten years of on-the-job experience since their residencies, and eighteen radiology residents in the first through fourth years of training diagnosed five chest X-ray pictures. We collected verbal protocols of the subjects in a modified version of the task that they normally perform in their offices. Ordinarily, a radiologist will examine a film or set of films and then dictate a formal report. In our task, we first had the subject look at the film for 2 seconds and report any abnormalities noticed. We then presented the film again without time limit. The subject was to think aloud while conducting an analysis and then to dictate a formal report. After this first formal report, we gave the subject some clinical data about the patient and then asked for a second analysis and report.

There was considerable variation in the conclusions that subjects reached about the films. In several cases, only a minority of the subjects had the correct diagnosis, and often the diagnoses varied in not only the disease suggested but even the anatomical structure thought

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This chapter was originally presented as a talk for the ONR Contractor Meeting at the University of California, San Diego on June 19, 1981.

Table 1.1
Final Dispositions of Target Feature of Film 8

Subjects	Final Dispositions
1st / 2nd Year Residents	
RA1	*No Interpretation* (description and call for additional information)
RA2	*Atelectasis* (right middle lobe *and* right lower lobe segment)
RA3	*Heart Configuration* or *Mass / tumor* or *Hilar Vasculature*
RA4	Calcified *Lymph Node* or *Pulmonary Artery*
RA5	*Heart Configuration*
RA6	*"Lesion"* (right middle lobe) *and Right Hilum* (summation shadow)
RA7	*Atelectasis* (right middle lobe)
RA8	*Heart Configuration* or *Pleural Thickening*
RA9	*Atelectasis* (right middle lobe) *and Pneumonia* (right lower lobe)
RA10	*Mass / tumor*
RA11	*Mass / tumor* or *Right Hilum* (unqualified)
3rd / 4th Year Residents	
RB1	*Hilar Vasculature* ("crowded")
RB2	*Mass / tumor* or *Pulmonary Sequestration*
RB3	*Esophagus* (dilated)
RB4	*Pulmonary Artery* (dilated)
RB5	*Mass / tumor* or *Esophagus*
RB6	*Atelectasis* (right middle lobe medical segment)
RB7	*Hilar Vasculature* (dilated)
Experts	
E1	*Atelectasis* (right lower lobe superior segment and right lower lobe medial basilar segment)
E2	*Atelectasis* (right middle lobe)
E3	*No Interpretation* (description and call for additional information)
E4	*Mass / tumor*
E5	*Mass*- like lesion; hypoplastic (small) lung area with compensatory hyper-expansion in other areas

to be the source of abnormal film appearance. Some of this variability can be seen in Table 1.1, which shows the range of final diagnoses for a film that was taken of a patient with a collapsed lung (or atelectasis). The area of collapsed lung appeared in the film as a triangular abnormality to the right of the heart. Figure 1.1 shows the film. There were several different, in fact quite divergent, diagnoses. These differences were of consequence, too. For example, tumors suggest general surgery, while atelectasis involves somewhat less invasive procedures in many cases, and abnormal vascular structure may require no action at all.

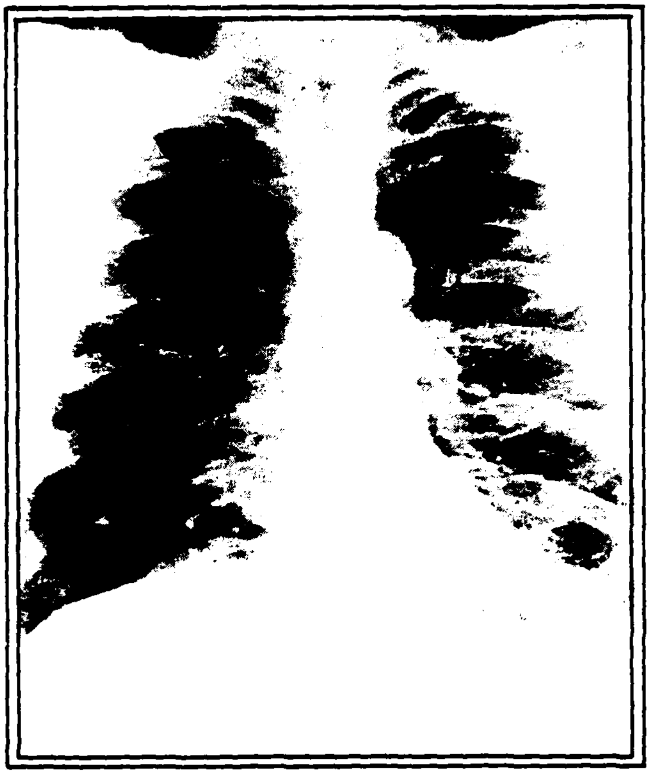

Figure 1.1 Film 8.

Table 1.2 shows four different types of formal reports for this film (Figure 1.1). The first response is the correct one, atelectasis. The second is noncommittal, the subject is ordering more films. The third response attributes the abnormality to abnormal vascularity, that is, blood vessels of abnormal size. The fourth subject has several alternative hypotheses: tumor, heart abnormality, and vascular irregularities. As is characteristic of all subjects on this film, the subjects all agree that there is an abnormality; they disagree only on its nature. Certainly, there are films that pose detection problems, because of low signal-to-noise ratio. However, we are not primarily interested in that aspect of the problem, but rather in the many cases in which film abnormalities are detected but misdiagnosed. Note also that the subjects vary in the amount and type of data that is in their final reports. Some have a specific description of the problem, some have extraneous comments that are not germane to the problem at hand, and some are too abstract or vague to be considered complete in their diagnosis.

Table 1.2
Four Examples of Formal Reports

Expert X

PA thorax. The lungs have the appearance of chronic obstructive lung disease and probably bullous emphysema. There is a well defined triangular density along the right cardiac margin, which in all probability represents atelectasis of the right middle lobe. I would like to see a right lateral chest in order to be certain of this determination.

Expert Y

The trachea is midline. The heart is not enlarged. There's a hyperlucency of the lung fields particularly on the right side which suggests the possibility of a hyperlucent lung etiology which is not apparent on this film. There is a density adjacent to the right heart border which should be further evaluated with other views, including oblique views and a lateral projection. There's no fluid at the bases. No free air beneath the diaphragm. Skeletal structures are essentially unremarkable. There's a nodular density noted in the right lung adjacent to the scapular angle which would also need further evaluation with tomography or other studies.

Resident I

My diagnostic report would be uh, a density involving the right hilar area and the right side of the heart, probably vascular.

Resident J

The costophrenic angles are clear bilaterally. There is increased markings of the lung fields bilaterally throughout their entirety. This is especially prominent on the left. These increased markings appear to be mainly vascular in origin. There's some suggestion of a reticular nodule pattern noted on the left lung field. The right lung fields have an unusual vascular pattern with large vessels extending out more peripherally than are usually seen. Uh, the cause of this, I'm not certain. There is blurring of the right hemidiaphragm probably caused by pericardial fat pad. The right heart border is not well visualized and a large well-marginated mass is seen in the lower mid-right paravertebral area This could conceivably be pulmonary outflow tract. A large tumor in this area is another possible etiology. I suppose that a very unusual cardiac configuration could give a similar picture. The heart is not enlarged. Bony elements of the thorax and soft tissues are unremarkable. Impression: increased pulmonary vascular markings, especially prominent on the left. The left hemithorax shows a slightly reticular, nodular pattern as well. There is a large right paravertebral mass of unknown etiology. Differential diagnosis would include a tumerous or cancerous mass, or unusual cardiac or vascular structure. There's no evidence of infiltrate or other similar active disease process.

Figure 1.2 Film 9.

Our initial efforts were to encode the protocols symbolically and to ask questions about the form of the encoded data: Did experts report more content (findings) from a film than novices did? How integrated were the reasoning steps in their explanations of those findings? How complex was a chain of explanations? There were differences between the experts and the novices that were revealed by such questions, but these differences were not sufficiently detailed to be the basis for any process modeling of the underlying differences in ability. Further, they did not distinguish between residents who were a year or two years apart in their training, even though a year may involve 10,000 or more film diagnoses (a large number of trials in conventional learning paradigms). Consequently, we conducted an in-depth qualitative analysis of the protocols, attempting to reconstruct the process of film diagnosis as completely as possible.

I.B ANATOMICAL REPRESENTATION ABILITY

One of the most striking differences revealed by this analysis was in the ability of more experienced subjects to see anatomy; that is, to build a rich mental representation of the anatomy of the patient whose radiograph is being diagnosed. Here is an example of the type of differences we found. On one of our films, shown in Figure 1.2, there was a small tumor in an area of the chest that contains many intertwined anatomical components (right suprahilar area). Two thirds of our first and second year residents and all of our advanced residents and experts detected abnormality in that region. However, only 28% of the residents who detected the abnormality and 60% of our experts correctly analyzed the problem as a tumor. There was a clear progression, with expertise, toward greater anatomical specificity in characterizing this abnormality. All five experts gave rich, specific, anatomical descriptions. For example:

> ..pulmonary hila themselves not enlarged...fullness in the mediastinum...a little above the hilum...not part of the aorta...definitely separate from the aorta...

However, even if we consider only those subjects who noticed abnormality, six of seven beginning residents and three of seven advanced residents failed to give clear, specific anatomical characterizations of the chest region containing the abnormality. They tended to speak of gross regions of the film rather than details of a representation of the patient's chest. This anatomical localization problem was also evident in the atelectasis film, Figure 1.1. There is no problem seeing an abnormal area; the issue is which organ it arises from. Table 1.3 shows the range of possibilities that arose in different subjects' interpretations. As you can see, the experts were pretty much in the right organ, at least, while the residents were seeing the problem as everything from an abnormal heart to an inflamed or diseased esophagus.*1

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*1 In a more recent experiment, in which we required a more immediate defense of such attributions, some of the incorrect attributions listed in Table 1.3 are never made, suggesting that it is robustness of anatomical knowledge that is at issue. When forced to consciously verify their conclusions, certain possibilities seem never to arise. This is not unlike certain children's arithmetic performance; they show bugs working on their own, but the bugs disappear when they do the problem with the teacher, even if the teacher provides no information.

There are three reasons for the difficulties that residents have in building and maintaining rich anatomical representations of the patient. First, they actually have not learned that much detailed spatial anatomy. That is, they do not have detailed, three-dimensional, Euclidean knowledge of the layout of organs in the body and of the range of variability in that layout. Most of the anatomy taught in medical school is systemic anatomy, the schematic knowledge of the routes between organs and organ components. In the last few years, a small amount of radiological (spatial) anatomy has been included, but not very much in light of the major complexities involved in converting from what is seen in a film to a three-dimensional representation of a patient's anatomy. Second, radiographic anatomy is particularly difficult, since a three-dimensional representation must be constructed from a two-dimensional projection that eliminates most depth information. There are no overlap cues for depth in an xray picture. The third source of difficulties is that the range of variation in anatomical structures is substantial. This variability often leads to situations in which a novice cannot decide, for example, whether an apparent density is part of a lung, part of the heart, part of an enlarged blood vessel, or a lymphatic tumor.

Table 1.3
Distribution of Anatomical Localizations over Different Organs (Film 8)

	Residents		
	Year 1,2	Year 3,4	Experts
Heart	26%	10%	8%
Lungs	35%	38%	85%
Non-lung Blood Vessels	19%	31%	0%
Lymph Nodes	7%	3%	0%
Pleurae	5%	3%	8%
Esophagus	7%	15%	0%
Right Bronchi	2%	0%	0%
	100%	100%	100%

I.C CONSTRAINT POSTING AND GLOBAL ENCODING

In addition to general differences in anatomical representation ability, we also found ability-related differences in the extent to which general diagnostic schemas were directly triggered by abnormality features. In many cases, experts detected a general pattern of disease that severely constrained the possible interpretations of otherwise ambiguous film abnormalities.

For example, in the collapsed lung film that gave rise to the protocols of Table 1.2, experts noticed very early that there were general film indicators of chronic obstructive lung disease. They discerned from textural indications in the lungs that the patient had emphysema, a sign of chronic lung disease. Patients with chronic obstructive lung disease tend to have smaller hearts, and (in some versions of the condition) the hilar area (where the blood vessels and windpipe plug into the lungs) tends to be smaller or less prominent than normal. The experts then appeared to keep in mind a series of constraints on their detailed interpretation of the film that were imposed by the knowledge that the patient had this condition. This phenomenon of constraint posting resulted in experts being less likely to misdiagnose constrained regions.

In the collapsed lung picture that we showed our subjects, the abnormal white area beside the heart could have been taken as a part of an extended heart or as an enlargement of hilar vascular structures, except for the constraints indicated by the emphysema that is evident in the rest of the lung tissue. Table 1.4 briefly summarizes the initial observations that subjects made about the heart and the right hilar area (the area near the heart where the windpipe and blood vessels plug into the lung).

Consider the top part of the table, which deals with comments made about the heart. A statement that the heart was small and/or shifted to the left is consistent with the constraints just mentioned. But, a statement that the heart was extended to the right and thus was the cause of the abnormal shadow is inconsistent with the constraints. There is a clear expert-novice difference evident. A similar contrast was evident when we considered interpretations of the right hilar area.

In general, this type of constraint posting is at a schematic level. A substantial schema, chronic obstructive lung disease in the present case, is triggered. It contains a number of separate constraints, some of which interact with other specifics of the case. For example, in more advanced stages of chronic obstructive lung disease, the heart <u>does</u> enlarge. So, if signs of <u>advanced</u> obstructive lung disease were present, the target abnormality might be considered appropriately as possibly being enlarged heart.

Table 1.4
Early Interpretations of the Heart and Right Hilum (Film 8)

	Residents					
	Year 1,2		Year 3,4		Experts	
Heart						
Small	18%	18%	14%	14%	40%	60%
Right-to-left shift	0%		14%		40%	
Normal	73%		71%		40%	
Source of target density	36%		43%		0%	
Right Hilum						
Decreased prominence	0%		14%		60%	60%
Medial displacement	0%		0%		40%	
Normal	73%		57%		0%	
Increased prominence or source of target density	45%		43%		0%	

One final note on constraint posting: Mark Stefik (1981), in his work on an expert computer system for designing genetic engineering experiments, has demonstrated the utility of early posting of constraints as a strategy for intelligent computer systems that solve complex problems. It is not surprising to see the same sort of strategy in a human expert. Our human experts also appear to post constraints in their working memories but to defer acting upon them until maximal data are available.

I.D THE INTERACTION OF THE COMPONENTS OF PROCESSING

In our analyses, we attempted to break down the diagnostic process into five major components and to examine the character and interactions of those components over the course of a diagnosis. The five components were:

- o <u>Film abnormality detection</u>. Noticing abnormalities in the X-ray film.

- Abnormality feature characterization. Generating a description of the abnormality, in terms of the perceptual features it contains.

- Anatomical localization. Modifying one's mental representation of the patient's anatomy to include changes in an organ that correspond to the film abnormality.

- Medical explanation. Explaining the anatomical variations in the mental representation.

- Overall case resolution. Completing an overall characterization of the patient in which the representation of anatomy and explanations of abnormality are as complete and consistent as possible.

There are several general conclusions we have made about the interaction of the anatomical localization and medical explanation components in the expert radiologist. They are presented below and then illustrated with an example protocol segment. The first conclusion is that developing a representation of the patient is more of a recursive process in experts. That is, initial film features are recognized as signs of abnormality. They, in turn, trigger schemas and post constraints that set the stage for further feature recognition. This recursion through the different stages of the diagnosis process is most successful and occurs in richer forms in experts. In diagnoses of the small tumor film shown in Figure 1.2, this stage-setting process was again quite evident. The experts were more likely to immediately see cues that prompted the triggering of a general schema for the chest, the chronic obstructive lung disease schema. Table 1.5 shows the evidence, from statements made after two seconds of looking at the film, for this schema having been triggered. Overinflated lungs, resultant low diaphragms, a small heart--all are indications of chronic obstructive lung disease. There is a clear expertise effect.

The expert has the ability to sustain the looking and reasoning cycle even in the face of considerable complexity. We currently think this recursive ability interacts strongly with the broad general schemas for constraining-syndrome information just discussed.

Another conclusion is that experts are opportunistic planners, in the sense discussed by Hayes-Roth and Hayes-Roth (1979), with very rich recognition and constructive perceptual abilities. They are very sensitive to new information and know when to seek additional data. We saw good examples in our protocols in which the whole course of reasoning in an expert was changed by the noticing of a single small anatomical detail. The residents were less likely to show this sort of behavior.

Table 1.5
Two-Second Encodings (Film 9)

Indication	Residents Year 1,2	Residents Year 3,4	Experts
Chronic obstructive lung disease or emphysema	18%	29%	80%
Hyperexpanded lungs	27%	57%	100%
Low diaphragms	18%	43%	100%
Small heart and/or narrow superior mediastinum	0%	29%	80%

Table 1.6
Final Dispositions for Subjects with Early COPD Schema (Film 9)

Disposition	Residents Year 1,2	Residents Year 3,4	Experts
COPD only	2/4	0/4	0/5
COPD - Hilar Vasculature	2/4	2/4	1/5*
COPD - Mediastinal Vasculature	0/4	1/4	1/5
COPD - Tumor	0/4	1/4	3/5

* Totally different argument than in residents.

For example, a lack of opportunistic processing in the residents was noted in the protocols for the small tumor film (Figure 1.2). Eventually, many of the residents noticed the chronic obstructive lung disease pattern. However, some persevered on that theme and used it to erroneously account for the density that was actually a tumor by deciding it was just enlarged blood vessels. Table 1.6 shows this effect. More of the residents who noticed the chronic obstructive lung disease syndrome failed to realize that one abnormality they noticed was a tumor unrelated to the overall lung condition. Table 1.7 shows some of the activity in one of our resident subjects as he fails to incorporate the possibility of a tumor into his chronic lung disease model and ends up not even mentioning it as a possibility.

Table 1.7
Example Protocol Segment

First Film Analysis

Okay - well there may be some asymmetry of the breast shadow, but I don't know if that's — it's probably within normal limits certainly both are present. Um, the heart is not enlarged although it may have a left ventricular contour. Uh, there's calcification in the aortic arch and probably some unfolding of the aorta. The mediastinum is not widened. *There is some prominence of the right hilar shadow - at least the inferior part of the hilar shadow I believe is all vascular and there is a suggestion of a mass in the right suprahilar region.* Uh, again there's a sort of a - well, there's a diffuse increase in the interstitial markings in both lungs - perhaps a little more accented at the bases, but not much difference between the bases and the apices. Um, no pleural effusions, - *there's hyperinflation of the lungs with flattening and scalloping of the diaphragm and therefore I'd raise the question of emphysema in this patient.* I don't see anything definite that looks like bullae or blebs although there is a suggestion of some small rounded densities on the left lower lung zone which may represent small blebs. I don't see any significant pleural reaction at the lung apices. Uh, the visible bones are markedly osteopenic - uh, I don't see any fractures - no pseudo-fractures in the clavicles. It's probably secondary to post-menopausal osteoporosis - OK.

First Formal Report

So the report - hyperinflation of the lungs and increased interstitial markings with prominence of the right hilar shadow which may be all vascular. These findings suggest chronic obstructive pulmonary disease. The heart is not enlarged and there's diffuse osteopenia.

Finally, our expert subjects were both more able to ignore irrelevant data and more able to take immediate account of relevant information. This was demonstrated in their responses to the clinical data they were given after making their first formal report. The following quotation shows the perseverance of a resident who had just been told that the very strange film he had seen was of a person currently in good health who had sustained lung surgery a decade earlier. The fact that the patient was now in good health seems not to have registered in the face of a complex explanation for the film that the subject had already constructed.

> ...there is no question that we are seeing evidence of heart enlargement as well as congested vessels and probable pleural effusion.... valvular disease, like mitral stenosis which can give you congested vessels as we see [here].... We also see evidence of interstitial pulmonary edema, which also

> goes with pleural effusion. .. We're seeing evidence of congestive heart failure.

These conclusions are illustrated by the protocol segment presented below. It is from an expert subject's first full examination of the film with a small tumor discussed earlier. This film, shown in Figure 1.2, like the collapsed lung film, showed evidence of emphysema. While the expert made an incorrect diagnosis, his protocol nicely illustrates several components of our emerging model. The subject, during the two-second viewing, activated a rich model of emphysema with broad implications for this condition, such as the expectation of a small heart. His initial model of emphysema was the classic version in which the hilar pulmonary arteries are prominent. He detected the shadow created by the tumor within two seconds and raised an incorrect, emphysema-driven interpretation for it that involved the hilar pulmonary arteries.

In his subsequent film analysis, after substantiating the emphysema (overinflation) in the lungs, the subject analyzed the pulmonary arteries and found his initial preconceptions in error. Direct perception of anatomical structure in detail prompted a change in hypothesis:

> ...The hilar structures are somewhat surprisingly small for the degree of overinflation in the lungs. This is an unusual finding as I've indicated because with the degree of overinflation one would expect much larger pulmonary arteries...

This caused a change in hypothesis to a different variant of emphysema, which involves <u>veins</u> instead of <u>arteries</u>:

> ...He seems to have pulmonary venous hypertension which is accounting for that area in the right supra hilar region [the tumor area] that I was uneasy about earlier on...

At this point, there is evidence for what artificial intelligence researchers call a <u>critic</u> process (Sussman, 1975). That is, there seemed to be an automatic realization that the previously posted constraint of small heart size was inconsistent with pulmonary venous hypertension:

> ...I'm trying to tie up this venous hypertension with the relatively non-enlarged heart...

The result of this criticism was a switch to yet another variant of emphysema, a version in which the disease was not uniform throughout the lungs:

> ...OK, the other explanation is that the diversion of blood flow to the upper pulmonary veins may be

> secondary to chronic lung parenchymal disease in the lower lobes--and that could be an alternative explanation. In that, he does not really have pulmonary venous hypertension, but he has diversion due to destructive lung disease in the lower lobes...

The subject maintained this specific theory variant through the first formal report on the film (given below) and all subsequent film analyses:

> ...There is pulmonary venous diversion to the upper lobes. This is in keeping with chronic obstructive airways disease which is most evident at both (lung) bases, but present, though to a lesser extent, in both upper lobes. Sorry, present to a lesser extent in the remainder of the lungs bilaterally...

Thus, the expert ended with an erroneous vascular interpretation for the feature in the film that was in fact produced by a tumor, which demonstrates that the schema-driven processing of experts is not always successful. In some instances, such expectations provide acuity in testing diagnosis hypotheses, and, in others, they prejudice the hypothesis testing process. The example also demonstrates some of the kinds of mechanisms available to temper excessive effects of initially triggered schemas, including automatic recognition of anatomical components, a rich substrate of medical and biological constraints for criticism, and a rich store of alternative interpretive frameworks.

To summarize, perceptual diagnosis is a complex, interactive process of constructing an interpretive theory of the patient shown in the film. The process opportunistically takes cues from the film (and other available sources of information), embellishes these from a rich medical knowledge base, criticizes and transforms these embellishments, and projects them back onto a representation of the patient's anatomy. This process recurs as new information is accumulated either through additional looking or through the internal products of diagnostic reasoning and building a mental representation of the patient's anatomy.

We are now at work building a model of this type of performance, based on the deeper analyses we have completed. The model will have several important features that are based upon our conclusions from the present study. First, it will have constraint posting that is produced by the triggering of relatively general schemas. Second, it will have to represent the differential adaptation of general schemas to the specifics of individual films. Third, it will have to account for the differential influence of posted constraints on experts and novices. Finally, it will have a recursive control structure, in which the stages of film abnormality detection, feature characterization, anatomical localization, medical explanation, and overall case resolution occur recurrently and opportunistically. While the model will deal specifically with radiology, we see it as a first step toward specifications for a family of models of diagnostic expertise and its acquisition. One long term goal of this work is an understanding of the respective roles of relatively general diagnostic tactics and specific diagnostic knowledge.

As noted above, this chapter is a summary of the work which is reported in the remainder of this report. In Chapter II, the overall rationale for this work is discussed, and relevant literature on radiological diagnosis is reviewed. Chapter III presents the details of the methods we have used thus far, and Chapter IV discusses a variety of preliminary analyses we conducted of the protocol data. Chapter V is the key chapter of the report, as it discusses the results of extensive in-depth analyses of the protocols we have gathered. That chapter is the source for the conclusions just presented.

CHAPTER II

RATIONALE

Our general focus is on the acquisition of skills which involve use of rich visual information to drive diagnostic decisions. Studying learning of such skills is important for several reasons. First, these skills are critical to many high-technology enterprises. Many diagnostic specialties, including equipment maintenance, medical diagnosis, and intelligence analysis, for example, require selective examination of large amounts of visually available information as part of the process of diagnosis (trouble-shooting) and treatment or repair. These specialties are among those for which recruiting and retention are difficult and training times are exceedingly long. Extending current theorizing on cognitive learning to this type of domain can have immediate applicability: Techniques can be derived for improving instruction in complex technical skills and suggestions can be made for machine assists that can replace some of the skills that take longest to learn. In addition, a theory of skill acquisition in realistic complex domains can be a strong basis for more general cognitive theories of learning.

The study of perceptually rich diagnostic domains is important for another reason. In such domains, the problem space is not clearly delimited. One quickly notices certain features but then has a choice between solving the diagnostic problem posed by the information seen so far or looking at the input picture, scene, or film for more information (which may in turn alter the problem definition). It is this feature that constrains the range of cognitive models which can be applied to such domains. It is also this feature of open-endedness that distinguishes these domains from other skill domains (such as reading) that are perceptually loaded but better constrained.

We have begun this work in the domain of radiology. It is a domain which meets the requirements of complexity, perceptual input importance, and open-endedness of data just discussed. Further, it is a domain in which even experts make many errors. Finally, we have been able to obtain adequate access to both expert and novice radiologists in sufficient number and under conditions conducive to the extensive analysis of performance we feel is required for us to progress toward our goals.

II.A THE RADIOLOGY TASK

We have been working exclusively with "PA chest" radiographs (chest x-ray pictures). Such radiographs are made by having a person stand facing and touching a 14 inch x 17 inch filmholder while an X-ray beam is directed at the film from a point behind (exactly 72 inches from the film) the person. "PA" stands for posterior-anterior and reflects the back-to-front movement of the beam through the patient. If the beam passes through radiotransparent material, such as air, it strikes the film and produces areas that are black after the film is developed. If the beam is refracted or absorbed, perhaps by organs or bone, fewer photons strike the film and such areas will appear clear or gray after development.

In contrast to normal pictures, in which the forwardmost objects in a scene occlude those further back, there are no overlap cues for depth in a radiograph. Rather, any region on a film is jointly determined by all tissues through which the beam passed in its trip from the X-ray source to the film plate. Bone, which blocks the photon beam, blocks out air and soft tissue whether the bone is in front of or behind the other tissue. There are slight depth clues due to the fact that the beam is a point source. Thus, the back portions of ribs appear slightly larger and fuzzier than the front portions, because they are further away from the film plate. However, for most purposes, there is not sufficient depth conveyed by such cues. For example, both front and side views are required to locate a bullet or tumor accurately. In certain cases, when several organs overlap in the two-dimensional film plane, distinguishing organ contours requires considerable expertise, a matter we will explore in more detail below.

II.B A SAMPLE PROBLEM

To further illustrate the nature of the radiologist's task and to introduce our experimental paradigm, we will discuss one of the films used in our study, the one shown in Figure 1.1 of Chapter I.

The major abnormality in this film is collapse (atelectasis) of the right middle lobe of the lung. The film presents both direct and indirect evidence for this condition. The direct evidence is a large, sail-shaped density along the right heart border. This density is the film's manifestation of the collapsed lobe itself. Inflated lung is full of radiolucent air, while collapsed lung is more radio-opaque, producing a whiter appearance in the radiograph. The indirect evidence is related to the causes and effects of lung lobe collapse.

There are three major causes for lung lobe atelectasis. <u>Obstruction atelectasis</u> results from deflation of a lung lobe as a consequence of obstruction in the bronchus (windpipe) supplying air to that lobe. In <u>contraction atelectasis</u> a lobe shrinks because of extensive fibrotic reaction to chronic inflammatory lung disease. Under compression atelectasis a lobe loses volume because of pressure from an adjacent structure that has enlarged. For example, a condition that can cause compression collapse in one lung lobe is emphysema in an adjacent lobe.

The film presents evidence of both emphysema and chronic fibrotic lung disease consistent with interpretations of compression or contraction atelectasis in a lobe. The most visible sign of emphysema is the presence of large dark regions in the lung. These dark regions are places where air pockets have been blown out beyond the normal air sac boundaries. They are darker than normal because the air is less radiolucent than the tissue it forces aside.

Also consistent with either atelectasis interpretation is the fact that the patient is elderly (70 yrs). In addition, the accompanying clinical data stated that there was no change between the present film and one taken a year earlier. Hence, the atelectasis is chronic rather than acute. This tends to favor a contraction interpretation since the causes of that condition are likely to be chronic. Obstruction atelectasis is perhaps more likely to be acute, for example, as a result of inhalation of a foreign object. Tumors would tend to produce compression or obstruction effects.

The above example case illustrates several aspects of the radiological diagnosis task. These are listed below:

- o There are several different places in the film which contain useful information.

- o There are multiple interpretations, even to the level of figure-ground distinctions, of apparent abnormalities. For example, some of our subjects saw the sail-shaped region as an addition of something new, a tumor, rather than as the subtraction of what would normally be expected at that point, substantial amounts of air.

- o Information from somewhat distinct abnormalities need to be combined (the air pocketing and the changed lung boundaries).

- o The interpretation of the radiographic evidence, and perhaps even initial perceptions, depend upon a detailed schema for the biophysics, normal and pathological, of the lung.

- o The problem environment presents a variety of constraints that may become applicable later in the diagnostic process.

- o Additional data arise, or are gatherable, throughout the course of looking at the film and reaching a diagnosis.

II.C PRIOR RESEARCH ON RADIOLOGICAL PERFORMANCE

The work we report represents a shift in the approach psychologists have taken to the study of radiological performance. Previous work has concentrated on the correctness of radiological diagnosis and, to some extent, on the overt behaviors of radiologists while looking at radiographs (Christensen, Dietz, Murray, & Moore, 1977; Garland, 1959; Gray, Taylor, & Hobbs; 1978; Kundel & LaFollette, 1972; Kundel, Nodine, & Carmody, 1978; Kundel, Revesz, & Toto, 1979; Kundel & Wright, 1969; Sandor & Swensson, 1978; Schreiber, 1963; Smith, van Belle, & Loop, 1978; Starr, Metz, Lusted, & Goodenough, 1975; Swensson, Hessel, & Herman, 1977, 1980; Taber, 1977; Thomas & Lansdown, 1963; Tuddenham & Calvert, 1961; Yerushalmy, 1969). This work has focused on radiograph reading as a signal detection problem, on environmental and training factors that affect diagnostic accuracy, and on eye movement patterns displayed while reading radiographs. The strongly accuracy-oriented concern of the existing literature is not surprising, since there are many types of diagnosis that are very difficult to make from simple standard radiographs, life-and-death decisions on which the accuracy of experts is considerably less than 100%. One reaction to this problem has been the continual effort of radiology researchers to produce improved imaging devices, such as the computer-generated axial tomogram. Another reaction has been to consider the problem as one of task analysis, task improvement, and training.

What has not been prevalent before now is research that is sensitive to the cognitive processing that goes on in the course of diagnosing a radiograph. Our goal in this project is to understand, to the point of being able to simulate it, the data gathering and decision-making processes that radiologists at various levels of expertise engage while doing their work. From this understanding, we hope there will flow ideas on how better to train experts in this domain and others.

While existing studies have not generally been process oriented, there are issues they raise that we need to consider. These issues are taken up in the brief literature review that follows.

II.C.1 Detection Studies

The difficulty of detecting abnormalities in radiographs is well documented. Garland (1959), in a review of detection studies to that time, estimated that radiologists fail to detect approximately thirty percent of abnormalities within films, and falsely detect abnormality where there is none in about two percent of films. These general rates have been shown to be highly robust (e.g., Tuddenham, 1962; Yerushalmy, 1969; Herman, Gerson, Hessel, Mayer, Watnick, Blesser, & Ozonoff, 1975). Much of the research into the causes of these failures has concentrated on physical characteristics of abnormal radiographic features as they influence detectability. In this regard, intrinsic feature characteristics such as size, contrast, and edge sharpness have been shown to influence the detectability of an abnormality (e.g. Goodenough, Rossman, & Lusted, 1973; Hallberg, Kelsey, & Briscoe, 1978; Kundel, 1981; Kundel, Revesz, & Toto, 1979; Sandor & Swenson, 1978).

However, various lines of research suggest that feature detectability is not confined to physical properties of the feature itself. Revesz, Kundel, and Graber (1974) demonstrated that the detection of a feature is partly dependent on the context in which it appears (see also Hallberg, Kelsey, & Briscoe, 1978; Kundel & Revesz, 1980; Revesz & Kundel, 1977). The importance of context has been further indicated by studies which have shown inferior detection when an observer's view is experimentally confined to the area of the abnormality (Carmody, Nodine, & Kundel, 1980; Swensson, Hessel, & Herman, 1978). Swensson (Swensson, Hessel, & Herman, 1980) has suggested such restriction produces its effects by preventing context dependent interrogation. For example, symmetric comparisons of a suspect portion of one lung to the same part of the other are not possible under this restriction.

Bearing on the role of context in detection is the observation by Revesz, Kundel, and Graber (1974) that what surrounds a given feature in a radiograph is largely structured rather than random or uniform noise; the context of an abnormality is a framework of anatomy. An abnormality represents either an addition to or a perturbation of this extant anatomical substrate (Ravin, 1980). An abnormality and its anatomical context can interact to camouflage the abnormality, to create emergent configural effects, or to create the appearance of normal variation within structures, etc. The role of structure in detection is further emphasized by studies showing that detection varies considerably with the anatomic location of an abnormality (Gray, Taylor, & Hobbs, 1978; Herman, Gerson, Hessel, Mayer, Watnick, Blesser, & Ozonoff, 1975; Smith, 1967). For example, Herman and his colleagues found that within the chest false-negative detections were particularly prevalent in the hilar regions while false-positive detections were prevalent in the pulmonary vasculature and lung parenchyma. These results prompted a proposal by Kundel and Revesz (1980) that detectability studies should supplement simple signal-to-noise measures of target features with measures that capture the meaning of the feature, as well as both the local and distant surround (see also Tuddenham, 1963).

Much of the work discussed above has emphasized the bottom-up influence of the film on the observer, but little has taken explicit account of the contributions made by the observer himself in the detection process. The need to appeal increasingly to context effects in order to account for detection suggests that the observer's evolving mental representation of normal and pathological anatomical structure do play an important role. Other observer functions which have been suggested include threshold for reporting detection (e.g. Swensson, Hessel, & Herman, 1977), judgements of the consequences of error as they affect detection (Kundel, 1981), integration of component radiographic features (Tuddenham, 1963), "set" influences (Blesser & Ozonoff, 1972; Tuddenham, 1962), and memory for experiences and radiographic patterns (Taber, 1977; Tuddenham, 1962). However, even these aspects are relatively superficial and do not tap ongoing cognitive processing in any detail.

To summarize, recent work on radiological diagnosis has suggested that studies of detection should take more explicit account of the observer. The approach we are taking is to directly study the observer by modeling the course of his cognitive processes rather than by introducing processing constraints into a detection model.

II.C.2 Search

Another area of previous research is the study of eye movements in the course of radiographic diagnosis. Studies in this area have found considerable interpersonal, intrapersonal, and interfilm variation in the overall scanning patterns of radiologists (e.g., Thomas & Lansdown, 1963; Tuddenham & Calvert, 1961). Indeed, Tuddenham found that the diagnostician in his study who had the most consistent and replicable film scanning pattern was also the poorest detector of abnormality (not inconsistent with our finding that the less skilled radiologists are less able to process opportunistically). However, to the extent that both the nature of the film and the diagnostic goals for the observer are specified in advance of reading, more stereotypic search strategies for a film can be demonstrated (Kundel & Wright, 1969). In addition, some investigators have found search strategy consistency to vary as a function of the experience of the diagnostician (Kundel, 1974; Kundel & LaFollette, 1972). But again, this consistency is film dependent. For normal chest films, there is an evolution, with experience, from a centrally oriented to a more circumferential pattern of initial scan. However, for abnormal films, search patterns are determined more by the information structure of the film (the pattern of abnormality) than by any general program of search.

Hence, Kundel (Kundel, Nodine, & Carmody, 1978) has described the search patterns of radiologists as being "neither random nor stereotyped." Some replicable characteristics of the search process have been demonstrated. For example, film coverage is non-uniform; much of the film is not fixated at all. Preference is given to borders and edges, while broad uniform areas tend to be excluded (e.g. Kundel & Wright, 1969; Thomas & Lansdown, 1963; Thomas, 1969). In addition, Kundel (1974) has recently demonstrated that much of search is focused on those film areas which radiologists assert are generally the most important sources of information relevant to the normalcy or abnormality of a film.

He concluded that the general distribution of coverage for a film is determined by a priori considerations but is adjusted to the particular content of the film. This claim is reinforced by the consistent demonstration that areas of abnormality, once detected, are refixated repeatedly (e.g. Thomas & Lansdown, 1963; Tuddenham & Calvert, 1961). Thomas has suggested that such refixation serves successively to build up an image (what we will call a representation) of a suspected abnormality (and perhaps its context). The diagnostician attempts to construct a meaningful representation of the film and is guided in this effort by prior knowledge of where important information is likely to lie.

This effort toward meaning is further suggested by two phenomena that appear in a number of reports in the literature. The first is that abnormal features are often fixated without being detected (Kundel & LaFollette, 1972; Taber, 1977). Taber (1977; see also Tuddenham, 1962) has suggested that this phenomenon stems partly from an inability to connect an abnormality with some internal memory structure that can give it meaning. A second observation is that when an interpretive framework for certain of the abnormalities of a film has been engaged, search tends to terminate and additional abnormalities are not detected (Christenson, Murray, Holland, Reynolds, Landay, & Moore, 1981; Smith, 1967; Taber, 1977; Tuddenham, 1965).

These considerations have led researchers to conceive of the film diagnosis process as being influenced by many sources. Sources of influence which have been proposed include perceptions of the film in the initial phases of diagnosis (Kundel & Nodine, 1975), prior knowledge of the patient (Schreiber, 1963), prior knowledge of the characteristics of films (Kundel, 1974), and the memory and interpretive experiences of the observer (Kundel & Wright, 1969), including the observer's memory store of interpretive models of anatomy, pathology, etc. (e.g. Taber, 1977, Tuddenham, 1962).

Radiologic diagnosis is seen as a multi-faceted process, involving elements of low-level sensation and perception along with higher-level cognitive processes. However, most models of the diagnostic process have treated these as largely linear and sequential stages, that is, processing proceeds from low to high level stages (e.g. Blesser & Ozonoff, 1972; Tuddenham, 1962). For example, Swensson (1980) has proposed a 2-stage stochastic model including detection, followed by decision-making (also referred to as cognitive evaluation). The work reported here is, in part, an attempt to elaborate on the processes that occur at different levels of the diagnostic process. It goes beyond earlier efforts in the number of components of processing considered, the qualitative detail with which they are characterized, and the extent to which component interactions are considered.

CHAPTER III

METHOD

We decided to begin our empirical efforts by conducting a study that was as ecologically valid as possible. Thus the task we chose was basically that of looking at a film and rendering a diagnosis. The task was modified to allow us to determine whether there were effects that were specific to the very fastest initial encodings, what the effects of receiving clinical data about the patient might be, and what sorts of reasoning went on prior to making a formal, for-the-record diagnosis.

III.A SUBJECTS

All of the subjects were physicians specializing in radiology. Eighteen were radiology residents in the local University teaching hospital system. Of these, eight were first-year residents, three were second-year residents, four were third-year, and three were fourth-year. One first-year resident, all third-year and one fourth-year resident had prior experience as physicians before starting the radiology residency. Except for one resident with five years of foreign practice, the others with experience had had a one-year internship. In addition to the 18 residents, five senior hospital staff radiologists participated, all of whom had ten or more years of post-residency experience. They ranged from 13 to 27 years of experience after residency (mean of 18.2 years) and estimated that they had analyzed in excess of 250,000 radiographs over the course of their training and medical practice. In contrast, no resident reported having experienced over 12,500 films. The residents were paid $5.00 per film and the staff radiologists were paid $15 per film for their diagnoses.

III.B STIMULI

Ten films were selected for use in this study. Each was a standard 14in x 17in PA (posterior-to-anterior, viz., patient facing toward the film plate, with the beam going through his back first) chest film. They were selected by the radiologist member of this research group (YW) in consultation with the other authors. The intent was that the films should range from normal through easy-to-diagnose abnormalities, to extremely difficult cases. In fact, all of the subjects were essentially correct in their diagnosis of the easiest film, and all but three were wrong on the hardest.

The experienced staff radiologists were only shown five of the ten films, due to limitations in the time they could make available for this study.

Table 3.1
Description of Films Used in Study

1.	Typical bronchopneumonia
*2.	Combination of metastatic lung tumors and infarction
*3.	Healthy patient who had partial lung lobectomy 15 years ago. Mediastinal structures shifted to occupy space formerly filled by excised lungs
4.	Normal chest; 45-year-old
5.	Large (4.5 cm) lung tumor plus less obvious mediastinal tumor and chronic lung disease
6.	Normal chest; 25-year-old female
7.	Chronic lung disease (fibrosis)
*8.	Atelectasis
*9.	Difficult-to-detect right, upper medial lung carcinoma
*10.	Right lower lung neoplasm and infarction; possible right hilar metastasis

*These films were seen by the five experienced radiologists.

Table 3.1 gives a brief description of each film. It also indicates which films the experienced radiologists saw. Films were presented on a portable viewbox of the sort common in physician's offices, and room lighting was adjusted with a dimmer to facilitate comfortable viewing.

III.C PROCEDURE

For each film, the following procedure was followed:

- The film was displayed for 2 sec.
- The subject was asked to report everything of interest that was noted during the 2-sec exposure.
- The subject was specifically prompted to report anything else of interest in each of the following areas in turn: lung fields including hila, diaphragm and pleural spaces, mediastinum, heart, skeletal structures, and anything else including other soft tissues.
- The film was shown again and kept visible while the subject was instructed to think out loud about what he was viewing.
- When ready, the subject dictated a final report on the film. This report was to be the sort of report that would have been made had the film been sent to the subject for reading in his everyday practice.
- A small amount of clinical data was shown to the subject. This consisted of a typed card with a sentence or two about the patient. For example, in the case discussed at the beginning of Chapter II, the following clinical data was provided: "CASE NO. 8: A 70 year old man who was admitted with unspecified G-I symptoms. A similar chest X-ray was noted about a year ago and shows no essential changes."
- The subject then looked at the film again, thinking out loud as before.
- When ready, a second formal report was dictated that took account of the clinical data that had been presented after the first report.

Both formal reports were dictated with the film remaining in view.

Prior to beginning the study, the subject was given an overview of the procedure and asked to sign a consent sheet. Each film's presentation took approximately five to ten minutes. The residents, in almost every case, spent about an hour in the study and returned on a second day shortly thereafter to complete the remaining films. The staff radiologists completed their five films in a single session. Each subject also completed a brief questionnaire concerning the nature of his current work, his medical experience and training, and an estimate of the approximate number of films to which he had been exposed.

III.D QUESTIONNAIRE DATA

Some of the results of the brief questionnaire were presented above in the description of subjects. A few additional points can be made. When asked to list the most common abnormalities they saw in radiographs, the residents and experts both reported lung problems most often. The second most prevalent class of abnormalities reported was heart problems for the residents and cancerous lesions for the experts. The subjects differed in the number of films they claimed to examine per day. As can be seen in Table 3.2, experts claimed to see more films per day. Four of the five experts claimed to see about fifty films per day and the fifth claimed to see about 140. *2

Table 3.2
Average Number of Films Seen per Day

Level of Training	Mean	s.d.
1st Year	41.6	20.7
2nd Year	40.0	10.0
3rd Year	38.8	11.1
4th Year	35.0	15.0
Expert	67.0	40.9

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*2 It is possible that some subjects interpreted this item as asking about the number of <u>cases</u> per day, even though the question referred to number of <u>films</u> per day.

CHAPTER IV

PRELIMINARY DESCRIPTIVE ANALYSES

We have undertaken two different approaches to scoring the protocols from this study. One approach, which will be treated in the next chapter, is an in-depth qualitative analysis of the nature of the reasoning process in the course of making a diagnosis. This approach uses a five-component model of radiological diagnosis as a framework for characterizing subjects' performance. This chapter describes a preliminary approach in which various quantitative analyses of the findings and explanations offered by subjects were performed without reference to any strong process assumptions.

The basic approach taken in this formal analysis was to analyze specific portions of the protocols, viz. the report after two seconds, the responses to the prompt questions after two seconds, and the two formal reports by listing all of the findings stated, along with any reasoning steps that connected them. These listings were then rendered into a standardized form and stored on the computer as a LISP data structure. *3

IV.A CHARACTERIZING A FINDING

It was necessary to find ways to assure that equivalent findings were encoded with the same symbol if they were effectively identical. The problem, of course, is to define what it means for two different verbal statements to be the same. This was handled in the following fashion. Each statement made about the film was encoded as a symbol that derived its name from the region or anatomical component being named, the property of that entity being discussed, and designators to indicate specifics such as left or right for organs like the lung and + or - for whether the property referred to was more or less prominent

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*3 LISP is a language specifically designed for manipulation of arbitrary symbolic data structures (see, for example, Meehan, 1979). It is the standard language for cognitive psychological simulations and has also been used for the most powerful computer-based medical diagnosis systems.

than usual. Care was taken to have different symbols for different levels of specificity. If any, the symbols were overspecific in differentiating between findings that were close to being paraphrases of one another. There were a total of 1586 symbols used for different findings and a total of 4498 findings reported altogether, summing over all subjects and all films, giving an average of 2.84 tokens (uses) for each type (unique code).

IV.B CHARACTERIZING A REASONING STEP

Reasoning steps were also recorded. Any statement in which one or more findings were claimed to be related to another finding or set of findings was scored as a reasoning step. These ranged from predictive statements such as "I notice engorged blood vessels in the hilar region, which suggests the possibility of congestive heart failure" to statements pointing out why a finding was unremarkable, such as "The aorta is tortuous but not surprisingly so given the age of the patient" to listings of several possible causes of an observed finding. Given a reasoning step that attributed Finding B as the result of Finding A, we refer to A as the cause side of the reasoning step and to B as the effect side. Both the cause side and the effect side of a reasoning step could have any one of three forms: a single finding, a conjunction of findings, or a disjunction of findings.

IV.C DEVELOPING THE DATABASE

All of the coded finding and reasoning step information was stored in a LISP data structure. This facilitated a variety of analyses of the data and will provide a basis for tying forthcoming modeling efforts to protocol data. The form of the data structure is quite simple. The LISP-encoded findings and reasoning steps of each subject on each film are structured into separate lists for each segment of each film. These lists are then represented as sets of properties associated with symbols which represent the entire performance of one subject on one film. These symbols, in turn, can be generated from a list of subjects or a list of films, so the entire dataset can be accessed in a straightforward manner.

A findings list is a simple LISP list with a symbol for each finding found in the given section of protocol. A reasoning step list is a list whose components represent individual reasoning steps. Each component is itself a list, with three elements: a type code, the cause side of a reasoning step and the effect side. The type code allows determination of the form of the cause side and the effect side (single finding, disjunctive list or conjunctive list). Each side is either a single finding or a list of findings, depending on the type code. Table 4.1 presents a piece of the protocol of one subject making a formal report on one film. Figure 4.1 shows the encoded representation of that

Table 4.1
Example Protocol Segment

Hyperlucency of the right lung compared to the left with extensive bullae formation in both right upper and right lower lobes. There is increased density along the right heart border with sharp borders which may represent atelectasis of the right middle lobe presumably secondary to the emphysematous changes as already described. There is loss of the medial portion of the – there's loss of the contour of the medial portion of the right hemidiaphragm and there may be atelectasis – segmental atelectasis in the right lower lobe as well. Again, presumably secondary to the previously mentioned bullae formation in the right lung. The left lung is normal without any evidence of infiltrate or tumor, the heart is of normal size and shape and without abnormality, the trachea is in the midline and there are no abnormalities of the soft tissues or bony thorax. Impression: Hyperlucency of the right lung with bullae formation and with secondary atelectasis of the right middle and segmental atelectasis of the right lower lobes.

film for that subject.

Associated with each finding is a an expanded verbal description of the finding and several properties. One property is the organ or region to which the finding refers. This enables easy access to all statements about a particular region or organ. Appendix C contains a printed expansion of the lisp-encoded data from one of the films (Film 8) for every subject.

The encoding may perhaps be better understood by a few examples. The phrase <u>hyperlucency of the right lung</u> was encoded by the symbol LUCLUNG+R, which had the expanded name INCR_LUCENCY_R_LUNG. The statement <u>there is increased density along the right heart border with sharp borders which may represent atelectasis of the right middle lobe</u> was encoded as (EXPL-- TRIDENSPERICR ATELOBEMIDR), which was expanded as:

```
TRIANGULAR/WEDGED/SHARP_DENS_R_HT_BORD/PERICARDIAL
     EXPLAINED BY
          R_MID_LOBE_ATELECTASIS
```

Expanded Printout

```
*** REPORT 1 ***
---     ---    0 — 0 INCR-LUCENCY-R-LUNG
CAUSE   EFFECT 0 — 0 BULLAE-R-UPPER-LOBE
CAUSE   EFFECT 0 — 0 BULLAE-R-LOWER-LOBE
---     EFFECT 0 — 0 TRIANGULAR/WEDGED/SHARP-DENS-R-HT-BORD/PERICARDIAL
CAUSE   EFFECT 0 — 0 R-MID-LOBE-ATELECTASIS
CAUSE   ---    0 — 0 EMPHYSEMA-R-UPPER-LOBE
CAUSE   ---    0 — 0 EMPHYSEMA-R-LOWER-LOBE
---     EFFECT 0 — 0 POORLY-SEEN-R-MEDIAL-DIAPHRAGM
CAUSE   EFFECT 0 — 0 R-LOWER-LOBE-ATELECTASIS

BULLAE-R-UPPER-LOBE
AND BULLAE-R-LOWER-LOBE
     EXPLAINED BY
          EMPHYSEMA-R-UPPER-LOBE
          AND EMPHYSEMA-R-LOWER-LOBE

R-MID-LOBE-ATELECTASIS
     EXPLAINED BY
          BULLAE-R-UPPER-LOBE
          AND BULLAE-R-LOWER-LOBE

R-LOWER-LOBE-ATELECTASIS
     EXPLAINED BY
          BULLAE-R-UPPER-LOBE
          AND BULLAE-R-LOWER-LOBE

TRIANGULAR/WEDGED/SHARP-DENS-R-HT-BORD/PERICARDIAL
     EXPLAINED BY
          R-MID-LOBE-ATELECTASIS

POORLEY-SEEN-R-MEDIAL-DIAPHRAGM
     EXPLAINED BY
          R-LOWER-LOBE-ATELECTASIS
```

Actual LISP Data Structure

```
(REPORT 1 (LUCLUNG+R BULLOBEUPR BULLOBELOWR TRIDENSPERICR ATELOBEMIDR
     EMPHLOBEUPR EMPHLOBELOWR POORSEEDIAPHMEDR ATELOBELOWR)
EXPLREPORT1 ( (EXPL** (BULLOBEUPR BULLOBELOWR)
                      (EMPHLOBEUPR EMPHLOBELOWR) )
              (EXPL-* ATELOBEMIDR (BULLOBEUPR BULLOBELOWR) )
              (EXPL-* ATELOBELOWR (BULLOBEUPR BULLOBELOWR) )
              (EXPL— TRIDENSPERICR ATELOBEMIDR)
              (EXPL— POORSEEDIAPHMEDR ATELOBELOWR) ) )
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Figure 4.1. Example formal coding of excerpt in Table 4.1.

IV.D RESULTS OF THE DESCRIPTIVE ANALYSIS

In analyzing the LISP-encoded protocols, we collapsed the subject sample into three groups: first and second year residents, third and fourth year residents, and experts. We then posed a number of quantitative questions about the data structures. Each of these questions involved counting the number of occurrences of a particular data pattern. Table 4.2 reports a number of such counts which, in every case are mean occurrences per subject per segment (two-second, prompt, first formal report and second formal report) per film. For each measure, a significant experience group effect was found ($p < .05$), and no interactions involving experience groups were found. The results for each measure are elaborated below.

Table 4.2
Comparisons between Experience Levels on Quantitative Protocol Measures

Measure	Residents 1st, 2nd Year	Residents 3rd, 4th Year	Experts
Number of causes*	2.56	2.58	3.62
Number of findings	6.58	6.63	9.09
Number of effects*	2.30	2.23	3.73
Longest reasoning chain	1.77	1.90	2.03
Biggest reasoning cluster	1.60	1.66	2.47
Percentage of connected findings	29.7%	29.6%	36.5%
Number of separate clusters	1.59	1.58	2.47

* By *causes* is meant findings that appeared on the cause side of at least one reasoning step; by *effects* is meant findings that appeared on the effect side of at least one reasoning step.

IV.D.1 Number Of Findings

The simplest question that can be posed is whether there are differences in the sheer number of findings reported by subjects of different levels of experience. Indeed, as can be seen in Table 4.2, experts reported more findings than did residents, and there were no differences in number of findings between the two resident groups. Whether we counted all findings, only those findings that were on the effect side of a reasoning step, or only those on the cause side of some reasoning step, the statistical effect was of about the same proportional magnitude.

IV.D.2 Properties Of Reasoning Structures

There are a variety of questions one can ask about the reasoning content of the protocols. The set of findings for a given subject on a given film can be thought of as a set of graph nodes which may be linked together by reasoning steps. A number of the properties of this graph are of interest. One can look for the longest path in the graph, one can ask what proportion of the nodes are interconnected, one can ask what the largest connected subset is, etc. We posed a number of these questions. In all of them, two nodes were considered to be linked if one of them was one of the findings in the cause side of a reasoning step and the other was one of the findings in the effect side of the same statement. Thus, if a statement was encoded as "A and B are explained by C and D," Then the following links would be recognized: AC, AD, BC, BD.

One question we asked in this graph analysis of the data structures was what the longest reasoning sequence was for each protocol segment for each subject on each film. The means for each experience group are reported in Table 4.2. This is the only statistic reported in Table 4.2 that shows a relatively continual progression of change over experience levels. The other statistics show only an expert-vs-resident effect. There seems to be a continual increase in the complexity of explanations and other reasoning steps that are offered by physicians for what they see in the films, as a function of expertise.

We posed additional questions designed to illustrate the extent to which expert protocols provided more integrated and complete explanatory reasoning about the abnormal features seen. One such question was, "What is the size (number of nodes or findings) of the largest connected subset in the protocol segment graphs?" Here, as can be seen from Table 4.2, there was again a difference between the experts and the residents. Similar differences were also observed for two other statistics, the proportion of findings mentioned in a protocol segment that were connected to the largest connected subgraph and the number of separate connected subgraphs for a single segment. As can be seen from Table 4.2, experts were both more integrated in their protocol statements and also more elaborative. That is, the primary cluster of explanations offered by an expert was likely to be larger than that of a resident, and the expert was more likely to offer additional, unconnected discussions as well.

There are several facts that must be borne in mind when examining these statistics. They are based upon all of the data, even though some of the reasoning steps may be incorrect. They also include both relevant and irrelevant findings and reasoning. It would be impossible to analyze only "correct" and "relevant" reasoning, since in many cases it is appropriate for a possible explanation to be posed at the outset but eliminated later on. The later eliminations are not always verbalized. There also may be reasoning links that the physician assumes are obvious and thus does not state overtly. In certain cases, this is obvious to the scorer as well, but in other cases, it is not. The result is an unknown amount of underestimation of the coherence of subjects' reasoning.

IV.E ACCURACY ANALYSES

We were interested in determining the accuracy and relevance of the various findings reported by the less experienced subjects. Therefore, the radiologist member of this group (YW) scored each finding reported for a given film on a nine point scale, on which low scores (1-3) stood for findings that were correct or justifiable and relevant to a diagnosis of the film, medium scores (4-6) stood for findings which were more or less correct but less relevant, and high scores (7-9) stood for findings which were incorrect, i.e., which did not accurately characterize the film. Because the acceptability of a finding for a film can change in the face of clinical data, each finding was given two scores, one based on no clinical data being available and one assuming the availability of the clinical data that was actually shown to subjects for the given film.

The accuracy data is shown in Table 4.3. The numbers in the table represent frequencies, per subject per film per protocol segment of findings at different levels of relevance. For each portion of the protocol, means are separately given for physicians in the first two years of residency, for more advanced residents, and for the entire sample. The overall conclusion we can draw is that the lack of strong differences in the numbers of findings reported at different levels of residency (see Table 4.2) is mirrored by a lack of strong differences in the accuracy and relevance of those findings that are reported. Another important finding is that the proportion of inaccurate statements of findings (i.e., statements of things that were not consistent with the film) was quite low, less than one wrong finding per protocol segment. Thus, even the early residents are generally not committing too many false positives of the sort that an expert would never commit.

Table 4.3
Classification of Residents' Findings by Relevance/Accuracy Scores
(Means over Films)

Protocol Segment	Relevance/Accuracy		
Year of Residency	High	Medium	Low
2 sec; Prompt			
1st, 2nd	1.37	2.07	0.87
3rd, 4th	1.61	2.78	0.77
COMBINED	1.46	2.35	0.83
First Report			
1st, 2nd	2.08	3.53	0.85
3rd, 4th	1.70	2.96	0.80
COMBINED	1.93	3.31	0.83
Final Report			
1st, 2nd	2.50	3.19	0.55
3rd, 4th	2.24	2.02	0.65
COMBINED	2.40	2.74	0.59

Table 4.3 also reveals an interaction between protocol segment and year of residency involving the number of stated findings. The third and fourth year residents stated more findings than the first and second year residents for the two-second interval, while the effect reverses for the final reports. We have no explanation for this result, other than a possible combination of changes in diagnosis with changes in expository style.

The results of the formal analyses suggest that two important aspects of the diagnostic process are the specification or noticing of relevant pieces of information and the reasoning processes that interconnect and help generate that information. For experts, more of the findings from a film elicit directional reasoning steps, and the resultant reasoning chains are longer and more interconnected. Clearly, though, a more detailed analysis of the protocols is needed if we are to understand the diagnostic process and how it differs in experts and journeymen. The next chapter describes the results of such deeper analyses.

CHAPTER V

IN-DEPTH ANALYSES OF THREE FILMS

In this chapter, we examine, in greater detail, the course of processing during the diagnosis of a film. Our analyses are organized around the set of five component processes that we proposed in Chapter I. This componential analysis is based on both the kinds of reasoning steps that were observed in the formal results reported above and a detailed re-examination of the protocols. We were especially sensitive to patterns of evidence that went beyond the boundaries of the individual protocol segments. Rather than independently scoring each utterance, we searched for patterns of explanation over the entire protocol for a subject on a film. Further, we looked explicitly for promising differences between subjects and reiterated our analyses to verify those differences.

V.A COMPONENTS OF INTERPRETATION

The following five process components have been useful in organizing our analysis. They should not, however, be thought of as stages in a bottom-up model of the diagnostic process; the sequencing of these components is only loosely constrained in the diagnostic process as we currently view it.

V.A.1 Abnormality Detection

In this component of the process, an abnormality in an x-ray film is noticed and judged worthy of further analysis. At this level, there is probably not yet a judgement regarding pathology but rather a perceived discrepancy between the visual pattern as presented in the film and some kind of schema for an idealized healthy chest. By this standard, even non-pathological consequences of aging (e.g., calcium loss in bones) would be noticed, but subsequently disposed of as "normal for a patient of this age" (a treatment of these features that is common in our protocols). We counted protocol content as evidence for abnormality detection if it contained discussion of the film area in which an abnormality was located or of specific components of anatomy existing in that area.

V.A.2 Abnormality Feature Characterization

Noteworthy abnormalities may be characterized with regard to their visual features. This characterization is in the language of shapes, sizes, and degrees of density, but, by definition, does not propose an underlying anatomical locus for the abnormality. An abnormality, for example, may be characterized as an "increased density" or a "stringy density" or a "large, well-circumscribed" or "diffuse" density. An example protocol excerpt illustrating feature characterization is given below:

> ...There's a peculiar semi-ovoid density against the right lower mediastinum. It has a sharp boundary to it. I'm not quite sure what it is yet...

V.A.3 Anatomical Localization

Anatomical localization involves identification of the anatomical locus of a target abnormality within the chest. An abnormality may be localized to an anatomical component (e.g., a fissure of the lung, the aorta) or to an anatomical region (e.g., "the right lower mediastinum" as in the example of feature characterization given above). The nature of the x-ray as a two-dimensional summation shadow of a collection of three dimensional anatomical components, and the associated lack of overlap cues, make anatomical localization a major challenge. The following example of an attempt at anatomical localization shows (a) the problem of anterior-posterior (heart to spine) localization, (b) the attempt to use auxiliary cues (the loss of borders) to assist in this determination and (c) the not uncommon call for additional chest views to help "triangulate" the source:

> ...I am not sure I can see the cardiac silhouette, which makes me think that this density may be adjacent to the heart, but it might also be related to the spine and further evaluation with oblique views or a lateral view of the chest would be helpful...

In the protocols given by subjects, a localization for an abnormality can be inferred directly—either as the placement for a feature characterization (see example under "Abnormality Feature Characterization" above) or in other direct attempts at localization (as in the example just given)--or indirectly through a medical explanation. For example, if an abnormality is interpreted as "lymphoma," this interpretation implicates the lymph nodes.

V.A.4 Medical Explanation

If an abnormality is assigned an anatomical source, a judgement can be made as to whether the source represents a pathological or a non-pathological condition. If a pathological condition is suspected, an attempt is made to determine an etiology or cause of the condition. For example, if an unusual density is localized to the heart, an interpretation may be made as to whether the heart, so appearing, is in some way pathological (e.g., a chamber enlargement) or is just an atypically configured but non-pathological heart. If the same density is localized to the right middle lobe of the lung, a judgement may be made as to whether it represents collapse or some form of infiltration in the lobe, etc. Interpretation may, in turn, result in the questioning of the original anatomical localization and an eventual decision to assign the abnormality to another organ.

V.A.5 Overall Case Resolution

Radiographic features do not exist in isolation but rather in a context of other features within the film and, when available, other information (e.g., history) about the patient. The overall case resolution component is the processing involved in attempts by the radiologist to reconcile a medical explanation either with the rest of the film ("internal" context resolution) or with other patient factors ("external" context resolution). Auxiliary features may serve to support an interpretation (underlined features in support of right middle lobe collapse in the first example below), to disconfirm an interpretation (the second example), or even to help choose among competing interpretations (the third):

> ...I <u>don't see the cardiac border</u> in this area. I see this triangular opacity that obscures the right border. So, now I really wonder if this right lung--right middle lobe--isn't collapsed. There's a <u>compensatory emphysema</u> (a common consequence of collapse) in the rest of the right lung...so I really wonder about lobar collapse...

> ...looking at the right lung and that density ("triangular," right heart border), a collapse of the lobe or lung can look like that, <u>but</u> I see lung markings fairly much throughout the lung with an exception of some paucity of markings in the right apex. So, I'm thinking that that is probably not a collapsed lung.

> ...So now I'm, I'm left with this triangular density. Okay. Do I see a minor fissure, for example? Is the minor fissure down? If the minor fissure's down, it will tell me that there is atelectasis as opposed to pneumonia. Or if there's pneumonia in a hypoventilatory component, the middle, the minor fissure would be

down...

Whether an explanation for an abnormality is maintained or abandoned depends on the subject's perceived degree of success in reconciling it with other available evidence. Again, the process of overall case resolution may result in a refinement or change in the localization for a target or even in a recharacterization of the target's features.

Although components of the x-ray reading skill have been proposed and discussed individually, we do not intend to minimize the interactions among them. On the contrary, the processes are highly interdependent. A particular way of characterizing what is seen may delimit or encourage various kinds of localizations and interpretations, and the process of testing an interpretation (case resolution) may modify a localization. Indeed, even from the examples presented, it can be seen that anatomical localization itself depends upon its own special subvariety of case resolution (e.g., in the use of borders and edges).

Also, since the schema(s) invoked to resolve a case may call for the presence of yet-unobserved features, it is reasonable to surmise that the resolution component triggers further activity in "earlier" components as well. The interactive dynamics of these components are illustrated in the analyses of protocols from three films that we used in our study. These analyses are presented next.

V.B FILM 8: CHRONIC RIGHT MIDDLE LOBE COLLAPSE

Overview. The remainder of this chapter presents analyses of subjects' performance on three of the films used in the study. Each film has its own contribution to make to an overall picture of the nature of the cognitive processes of diagnosis. Film 9 involved a single target abnormality, with no structurally related abnormalities. Film 8, also contained a single but more salient abnormality. However, in contrast to Film 9, Film 8 contained abnormal manifestations throughout the chest that were structurally related to the target abnormality. Film 2, represents yet a different type of film. Film 2 contains multiple features of abnormality, most of which are readily apparent, and there is no predominant main abnormality. The etiological relationships among these abnormalities are the primary problems to be resolved in diagnosis of the film.

V.B.1 Overview Of The Task For Film 8

The major abnormality in this film (shown in Figure 1.1 above) is chronic collapse (atelectasis) of the right middle lobe of the lung. The film presents both direct and indirect evidence for this condition. The direct evidence is a large, sail-shaped density along the right heart border. This density is the film's manifestation of the collapsed lobe itself. The indirect evidence is related to the causes and effects of lobar collapse (see earlier discussion in Section II.B). The most important of these auxiliary features are evidence of hyperexpansion (increased air volume) in the other two lobes of the right lung, and chronic fibrotic disease. Under an obstruction or contraction atelectasis interpretation, hyperexpansion would be an expected effect of atelectasis, a compensatory result of the volume loss in the right middle lobe. The collapse of the lobe itself is the result of some kind of obstruction of air flow to the lobe, or the fibrotic changes in the lung. Under an interpretation of compression atelectasis, the hyperexpanded lungs <u>cause</u> the collapse in the middle lobe by the increased pressure they exert on it, and the expansion itself results from some other independent source of air trapping (e.g., emphysema).

The clinical information we presented states that the patient is elderly (70 yrs.) and that the film shows no changes from one taken a year earlier. This information is easy to reconcile with a compression or contraction interpretation for the atelectasis since the causes of these conditions would be expected to develop and be stable over long periods of time. Obstructive atelectasis is harder to reconcile with the lack of change in the film since many causes of obstruction (e.g., tumor impinging on the bronchus which supplies air to the lobe) might be expected to show faster change.

In an idealized, successful analysis of the film, a physician would notice the abnormality on the right heart border, characterize it as sharp bordered or "triangular," and localize it to the right middle lobe of the lung. He would further interpret the abnormality as pathological and as resulting from collapse. Furthermore, he would reconcile this interpretation with the age of the man and with the convergent evidence in the film: hyperexpansion and chronic fibrotic lung disease consistent with compression or contraction collapse. The idealization just presented does not fully capture the extent of processing to be expected from an expert. There are a number of other disease schemas that might be triggered and would then have to be rejected on the basis of complex detail in the film.

V.B.2 Overview Of Subject's Performance

Of the twenty-three subjects who read Film 8, only fourteen (61%) ever considered an interpretation of atelectasis during analysis of the film, and only five (22%) maintained this interpretation in their final reports for the film. This is despite the fact that all subjects "detected" the abnormal density along the right heart border and judged it as requiring further explanation. Hence, although detection of the main abnormality was not a major issue on this film, explanation of this abnormality posed serious difficulties for many subjects. The protocol data is discussed with respect to each of the five component processes in turn, below.

V.B.2.a Detection -

All but two subjects detected the target abnormality within the initial two-second viewing period. This suggests that the target feature in this film is visually salient. The only subjects who did not evidence detection of the target in two seconds were experts (E1, E4). A possible explanation for the lack of quick detection among experts on this film will be proposed later in this chapter.

V.B.2.b Final Dispositions -

Before discussing the other four components of processing, we consider the final dispositions subjects made in this case. These final dispositions provide useful clues for understanding protocol phenomena that correspond to the different components of the process. By final disposition for an abnormality, we mean the ultimate fate of that abnormality within the subject's overall analysis of the film. (Recall that the procedure used in the study involved five reporting periods--a two-second viewing and report, two unconstrained periods of analysis, and two formal reports.) For various reasons, the final disposition is not necessarily the final explanation for the abnormality given in the subject's last formal report.

For example, a subject might have detected an abnormality early (perhaps in the first full viewing), attributed it to some benign or normal condition (e.g., a vascular structure), and never mentioned it again. Another subject might have detected and explained an abnormality early in the diagnosis process and then abandoned the explanation for a more general etiological hypothesis that subsumes that abnormality along with other features of the film. Subsequent analyses may have focused on these related conditions and completely ignored the original abnormality. Still other variants of final disposition exist, including simple description of the visual properties of an abnormality without interpretation.

Table 5.1
Final Dispositions of Target Feature on Film 8

Subjects	Final Dispositions
1st / 2nd Year Residents	
RA1	*No Interpretation* (description and call for additional information)
RA2	*Atelectasis* (right middle lobe *and* right lower lobe segment)
RA3	*Heart Configuration* or *Mass / tumor* or *Hilar Vasculature*
RA4	Calcified *Lymph Node* or *Pulmonary Artery*
RA5	*Heart Configuration*
RA6	*"Lesion"* (right middle lobe) *and Right Hilum* (summation shadow)
RA7	*Atelectasis* (right middle lobe)
RA8	*Heart Configuration* or *Pleural Thickening*
RA9	*Atelectasis* (right middle lobe) *and Pneumonia* (right lower lobe)
RA10	*Mass / tumor*
RA11	*Mass / tumor* or *Right Hilum* (unqualified)
3rd / 4th Year Residents	
RB1	*Hilar Vasculature* ("crowded")
RB2	*Mass / tumor* or *Pulmonary Sequestration*
RB3	*Esophagus* (dilated)
RB4	*Pulmonary Artery* (dilated)
RB5	*Mass / tumor* or *Esophagus*
RB6	*Atelectasis* (right middle lobe medical segment)
RB7	*Hilar Vasculature* (dilated)
Experts	
E1	*Atelectasis* (right lower lobe superior segment and right lower lobe medial basilar segment)
E2	*Atelectasis* (right middle lobe)
E3	*No Interpretation* (description and call for additional information)
E4	*Mass / tumor*
E5	*Mass-* like lesion; hypoplastic (small) lung area with compensatory hyper-expansion in other areas

Table 5.1 gives the final dispositions of all subjects for the target abnormality of Film 8—the density on the right heart border reflecting right middle lobe collapse. These can be used to gain an understanding of the major alternative interpretations for the key feature in the film. To this end, Table 5.2 gives the information of Table 5.1 in a different form, showing the different subjects who utilized each interpretation in their final dispositions and percentages of subjects who included each interpretation.

Table 5.2
Final Dispositions of Target Feature on Film 8 (retabulated)

	Residents (n = 11)		Residents (n = 7)		Experts (n = 5)		
Interpretations	1st, 2nd Year	%	3rd, 4th Year	%		%	% Total
Atelectasis	RA2, RA7, RA9	27	RB6	14	E1, E2	40	26
Mass / tumor	RA3, RA10, RA11	27	RB2, RB5	29	E4, E5*	40	30
Vascularity (dilated, normal, etc.)	RA3, RA4, RA6, RA11	36	RB1, RB4, RB7	43			30
Heart Configuration	RA3, RA5, RA8	27					13
Esophagus (dilated, unqualified)			RB3, RB5	29			9
Pleural Thickening	RA8	9					4
Calcified Lymph Node	RA4	9					4
Pneumonia	RA9	9					4
Pulmonary Sequestration			RB2	14			4
Lesion (right middle lobe)	RA6	9					4
No Interpretation	RA1	9			E3	20	9

Note. Percentages do not add to 100% because of conjunctive and disjunctive interpretations.
* Includes hypoplasia in a right lung area.

While there were a number of different final dispositions, they fell into five major categories, some of which were important only within particular subject groups. Each of these categories is considered below.

V.B.2.b.i Atelectasis -

This is a collapse argument. Subjects offered different locations for the collapse, ranging from the right middle lobe to other lobes or lobe segments. Only six (26%) subjects concluded atelectasis of any kind.*4 Five of these attributed some involvement to the right middle lobe but only two (RA7, E2) gave what we considered to be the "correct" interpretation: collapse of the entire middle lobe and only the middle lobe without involvement of additional processes such as pneumonia. (It should be noted that involvement of additional processes, such as pneumonia can be ruled out, but only on the basis of relatively subtle details.)

V.B.2.b.ii Mass/tumor -

Despite slightly different forms (e.g., tumor, carcinoma, benign cyst), this is basically an argument that the target abnormality is a "growth," something that has developed in the target area that should not be there. Mass/tumor was a major competitor as an explanation of the target abnormality for many subjects and within all subject groups. It was a final disposition for seven of them (30%), in equal proportions at each experience level.

V.B.2.b.iii Vascularity -

This argument attributes the target abnormality in some way to the major vessels in the area of the right hilum. Subjects were more or less specific about which vessels they considered to be involved (e.g., "hilar vasculature," "right pulmonary artery") and about the particular condition of these vessels (e.g., engorged, "crowded," unqualified as to condition). Vascularity was a major final interpretation for residents but not the experts. Indeed, vascularity was the <u>modal</u> interpretation in both resident groups (see Table 5.2) but was offered by no expert.

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*4 This number is different from others given in this report because it refers to <u>final dispositions</u> rather than <u>final reports</u>.

V.B.2.b.iv Heart Configuration -

This argument attributes the target abnormality to the heart itself. The heart is construed as "unusually shaped" or in an "unusual position" or perhaps completely normal. What is important is that the heart is seen as the source of the target shadow, rather than something adjacent to the heart or in the area of the heart. A heart interpretation was concluded only by 1st and 2nd year residents but was a final disposition for three in this group (27%).

V.B.2.b.v Esophagus -

These interpretations attribute the target density to the esophagus. For subjects including it as a final disposition, the esophagus was claimed to be dilated or was unqualified as to condition. Esophageal arguments were offered by only two subjects, both in the third and fourth year residents' group.

V.B.2.b.vi Summary Of Final Dispositions -

The preceding discussion has shown that Film 8 is a film in which the great majority of subjects reached erroneous conclusions. Yet, alternatives to the basically correct interpretation (some sort of atelectasis determination) clustered into four groups. Mass or tumor interpretations were a major foil for all three experience groups and were the only non-atelectasis conclusions given by experts. There were three other alternatives offered by the pool of residents. Both more and less experienced residents were drawn to arguments involving vascularity. Two 3rd and 4th year residents reached an esophageal interpretation, and a heart interpretation was concluded by three first and second year residents.

V.B.2.c Abnormality Feature Characterization -

The performance of subjects on Film 8 suggests that feature characterizations influenced both successful and unsuccessful explanations. Across all subjects and the five reporting periods (two-second, first free view, first report, second free view, second report), the probability of a subject's raising atelectasis as an interpretation for the target abnormality was higher in protocol segments in which it was characterized as "triangular," "wedge-like," or "sharp margined," etc. (0.48, i.e., in 16 of the 33 reporting period protocols for which subjects characterized it as "triangular", they raised the possibility of atelectasis) than when it was characterized, but characterized in some other way, e.g., unqualified "increased density," or "irregular density" (0.28, or 15 out of 54).

Within reporting periods where the abnormality was characterized as non-triangular, the probability of an atelectasis interpretation was fairly uniform across subject groups (0.26 for 1st and 2nd year residents, or 7 out of 27; 0.29 for 3rd and 4th year residents, or 5 out of 17; and 0.30 for our experts, 3 out of 10). Within periods where it was characterized as basically triangular, the tendency to advance an atelectasis interpretation was perhaps greater among the experts (six out of seven occasions or 0.86 for experts, compared to 0.39, or seven out of 18 for 1st and 2nd year residents and 0.38, or three out of eight, for 3rd and 4th year residents).

While these results can only be suggestive (the numbers are small and non-independent), they may shed some light on the role of abnormality feature characterization in the film reading process. In particular, labeling (characterization) of an abnormality's visual features may affect its likelihood of eliciting (triggering), or being judged consistent with, various interpretation schemas. For example, if a density is seen as "sharp margined" it may be more likely to elicit an interpretation related to anatomic deformity, whereas if it is seen as "diffuse" or "fuzzy," it may more likely trigger interpretations which are less confined to anatomical boundaries (e.g., infiltration, effusions). By this argument, the "same" abnormality can be "seen" in different ways, depending upon which of its features are verbally characterized. These characterizations, in turn, favor some lines of further interpretation rather than others.

V.B.2.d Anatomical Source Localization -

Our analyses of anatomical localization are concerned with the use of anatomical knowledge to construct a mental representation of the anatomy that underlies a target film abnormality. We used several types of evidence in these analyses. First, subjects often characterized abnormalities in anatomical terms (e.g., "increased density in the hilar vessels"). Second, subjects may have overtly discussed specific anatomical components as the locus of an abnormality (e.g., "definitely separate from the aorta, hence probably involves the pulmonary arteries"). In these instances, anatomy can be used inclusively (as the placement for the abnormality), exclusively (to localize by excluding alternatives), or to establish points of reference. Finally, some medical explanations offered for an abnormality may indirectly implicate an anatomical location. For example, if a subject interprets an abnormality as a "bronchogenic carcinoma," this is evidence for a localization to the bronchi. Localizations reported in this section refer to anatomy used in any of the ways discussed above.

Table 5.3 shows the relative frequency distribution of anatomical localizations offered for the main abnormality of this film (which corresponded to the collapsed lung).*5 Statements suggesting localizations were counted separately for a subject if they involved (1) the same component (e.g., heart) in two different reporting periods

Table 5.3
Distribution of Anatomical Localizations over Different Organs (Film 8)

	Residents		
	Year 1,2	Year 3,4	Experts
Heart	26%	10%	8%
Lungs	35%	38%	85%
Non-lung Blood Vessels	19%	31%	0%
Lymph Nodes	7%	3%	0%
Pleurae	5%	3%	8%
Esophagus	7%	15%	0%
Right Bronchi	2%	0%	0%
	100%	100%	100%

or (2) different components (e.g., heart, lung) within the same reporting period. Hence, a subject who localized the abnormality as part of the heart in two different reporting periods would contribute twice to his group's heart count and twice to the total of group localizations.

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*5 Regional localizations are not included within this table. Several large areas of a film serve dual roles, both as gross anatomical components and as major regional demarcations of the visual plane. Included among these are the lungs, hila, and mediastinum. For a localization to the lungs, for example, to be included in Table 5.3, there had to be indication that the subject was using the lung anatomically (e.g., reference to lobes or segments) rather than, essentially, as the right side of the chest.

Most expert anatomical localizations for this film were concentrated in the lung (11 of 13, 85% of total separate localizations).*6 One expert mentioned pleura as an anatomical location, but actually in a way that might include the lung ("pleural tenting"), while the one expert who mentioned the heart did so to exclude it ("separate from the heart").

In contrast, within resident groups, it appears that (in addition to the lung) the heart, various vessels in the area of the target feature, and the esophagus played a more substantial role in anatomical localization of the abnormality. The heart was, perhaps, more important to the 1st and 2nd year residents while the esophagus was more important to the 3rd and 4th year residents.

The anatomical localizations for Film 8 suggest that incorrectly isolating the affected anatomy contributed substantially to difficulties that residents experienced with the film while this was not a major problem spot for the experts. As discussed next, this account of the results is reflected in the medical explanations proposed by subjects.

V.B.2.e Medical Explanation -

Medical explanation is the component of processing which goes beyond anatomical localization to propose a state or condition of an anatomical component or chest region which could account for an abnormality in a film. Interpretations can range from judgements of normalcy (e.g., normal pulmonary artery), to pathophysiological conditions (e.g., engorged pulmonary artery) and disease processes affecting an area (e.g., pneumonia). Hence, while an anatomical component may be referenced in localization of a abnormality without attributing that abnormality directly to the component (e.g., "separate from the heart"), a medical explanation involving the same component must more directly implicate it somehow as a cause for the appearance of the abnormality on the film.

Table 5.4 gives the kinds of explanations that subjects raised for the target abnormality of Film 8 and the particular subjects who raised each explanation during their analyses of the film.*7 Subjects were counted as having considered an explanation for the abnormality if they raised it even once in the course of their entire diagnosis.

The results shown in Table 5.4 reaffirm findings we have already discussed. The five major arguments (atelectasis, mass, vascularity, heart, esophagus) included among subject's final dispositions (Table 5.2 and related discussion) are substantiated as the major explanations subjects investigated during the course of their film analyses. Comparing Tables 5.4 and 5.2, more subjects are seen to have considered some arguments than to have persevered with them. In particular, the heart-related explanations which were absent from the final dispositions of 3rd and 4th year residents are seen to have been considered by 3 of 7 of these subjects (43%).

Table 5.4
Subjects Who Considered Various Interpretations for the Target Abnormality of Film 8

	Residents		Residents				
Interpretations	1st, 2nd Year	% (of 11)	3rd, 4th Year	% (of 7)	Experts	% (of 5)	% Total (of 23)
Atelectasis	RA1, RA2, RA4, RA7, RA9	45	RB2, RB3, RB6, RB7	58	E1, E2, E3, E4, E5	100	61
Mass / tumor	RA1, RA3, RA4, RA8, RA10, RA11	55	RB2, RB3, RB5, RB6, RB7	71	E2, E4, E5	60	61
Vascularity (dilated, hypertension, crowded, normal, etc.)	RA1, RA3, RA4, RA6, RA7, RA11	55	RB1, RB4, RB5, RB7	58			43
Heart Configuration	RA3, RA4, RA5, RA8, RA11	45	RB4, RB6, RB7	43			35
Esophageal (dilated, stricture, hiatal hernia)	RA9	9	RB2, RB3, RB5	43			17
Pleural (think, effusion, tenting)	RA7, RA8	18			E3	20	13
Lymphatic (calcification, adenopathy)	RA4	9	RB5	14			9
Pneumonia (infiltrate)	RA9	9	RB7	14	E2	20	13
Pulmonary Sequestration	RA5	9	RB2, RB5	29			13
Lesion (right middle lobe)	RA6	9	RB7	14	E2	20	13

It is clear from Table 5.4 that among expert subjects the major problem posed by the film was one of medical explanation rather than anatomical localization or abnormality detection. Except for one interpretation associated with the pleura, all expert interpretations were related to the lung. Every expert subject raised an atelectatic interpretation and the only major competitor to atelectasis was mass or tumor. No expert subject ever suggested an explanation associated with vascular structures, the heart, or the esophagus. In contrast to the experts, it is clear that among resident groups, Film 8 posed possibly interactive problems of medical explanation <u>and</u> anatomical localization.

Another noteworthy feature of Table 5.4, when compared to Table 5.2, is the high rate of ultimate rejection for atelectasis explanations, which would have been correct. While atelectasis was considered by 61% of subjects (including all experts), it was the final disposition for only 26%. In addition, the rate of rejection was high among all subject groups (2 of 5 first and second year residents who had considered atelectasis; 3 of 4 third and fourth year residents; 3 of 5 expert).

Two questions are raised by the results discussed in this section:

- o Why were the experts so thoroughly able to avoid the particular erroneous arguments (except "mass") that were frequently raised by the residents?

- o Why did so many subjects ultimately rejected atelectasis?

The next two sections will address these questions.

V.B.3 Mechanisms Of Expert Selectivity

The discussion thus far has suggested that experts were more selective in their anatomical localizations and medical explanations than were residents. <u>This section considers possible mechanisms for</u>

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*6 The numbers are small but, if anything, generally underestimate the involvement of each anatomical component: Multiple mentions of the same component within a single reporting period were treated as one localization. This included periods in which a subject referred to different subcomponents (e.g., different lung lobes or different heart chambers) of the anatomy captured at the level of the table headings.

*7 Table 5.4 included only explanations for the specific target abnormality that were based upon anatomical localization to an area specified in Table 5.3. Very few interpretations outside this set were ever raised by subjects.

this selectivity among the experts. Through this analysis, the role of overall case resolution, the fifth of the components proposed above, also becomes apparent. Subjects' initial analyses of the heart and right hilum provide insight into the mechanism by which experts were able to avoid these potential cul-de-sacs in the diagnosis of Film 8. Table 5.5 (which also appeared earlier as Table 1.4) shows the frequencies of medical explanations offered by subjects during the initial two-second viewing period and immediately thereafter during the first analysis of the film.

Table 5.5
Early Interpretations of the Heart and Right Hilum (Film 8)

	Residents					
	Year 1,2		Year 3,4		Experts	
Heart						
Small	18%	18%	14%	14%	40%	60%
Right-to-left shift	0%		14%		40%	
Normal	73%		71%		40%	
Source of target density	36%		43%		0%	
Right Hilum						
Decreased prominence	0%		14%		60%	60%
Medial displacement	0%		0%		40%	
Normal	73%		57%		0%	
Increased prominence or source of target density	45%		43%		0%	

Three of the five experts immediately saw the heart as either unusually "small" or displaced leftward so that the right heart border (in the area of the target density) was lost within the spinal column.*8 In contrast, the great majority of residents either saw the heart as normal or had already engaged the heart as at least a possible anatomical locus for the target abnormality. With regard to the right hilum, 3 of 5 experts judged the right hilum to be reduced in prominence, less well seen than usual. Among explanations given for

*8 Note that Expert E3 does not show the same patterns as the other experts. In fact, the assessment of the heart offered by the other experts was not articulated by expert E3 until the second free viewing period.

this was that the hilar vasculature was displaced medially (toward the mediastinum or center of the chest) and, as explained by E1, largely "hidden" behind the mediastinum. Again, nearly all residents judged the right hilum to be either normally prominent or even increased in prominence, and some residents thought that the right hilar vasculature was the source of our target density.

Experts' early assessments of the heart and right hilum would make it very difficult for them to later attribute the target density to these anatomical components. In particular, a view of the heart as hypoplastic (unusually small) would be largely incompatible with a claim that the target density in the film is <u>composed of</u> heart structure. For the target density to <u>be</u> heart, the right heart border would have to extend farther to the right of the spine than is even usually the case; that is, this interpretation would probably require at least local "enlargement" or protrusion of the heart. Similarly, assessments of the right hilar structures as diminished in prominence and specific "out-of-the-way" placements of major hilar vessels would largely preclude subjects from eventually attributing the target density to hilar structure, as shown in this protocol excerpt:

> E1:...The right hilar vessel is hidden behind the mediastinum and lower than expected so we don't see clear right hilar vessels...

The role played by early schema activation in later anatomical localization is an important example of the interactive (recursive) flow of control between the five components we have identified.

Experts' protocols established various correspondences between their early assessments of the heart and hilar vasculature and more global dynamics of Film 8 involving chronic obstructive pulmonary disease e.g.:

> E2:...The heart is so hypoplastic in appearance that it doesn't really project beyond the right side of the spine. And, this is the type of heart you see in chronic obstructive pulmonary disease...

> E1:...The right lung is larger and more lucent particularly in the region of the right middle lobe and right lower lobe, quite consistent with rather diffuse emphysema. Because of that, the right hilar vessel is a little bit smaller than the left hilar vessels...

<u>Herein lies an explanation for the expert selectivity demonstrated on the film.</u> Experts may have encoded or quickly classified the chest (the film) according to its major, global physiological dynamics--i.e., chronic obstructive lung disease. A global assessment of this kind might, in turn, carry severe restrictions for the later analysis of more "local" aspects of the film--a process involving "constraint posting"

(Stefik, 1981a,b). Early global encoding by experts may, in turn, account for the curious fact that only experts failed to detect the target density on Film 8 during the initial two-second viewing period. This phenomenon will be taken up again in discussion of the next film where it is, perhaps, even more clearly evident.

We still need to explain why the experts were able to exclude the esophagus as a possible anatomical locus for the abnormality. The clinical data we provided for the film at the second full analysis contained a coincidental statement that the patient had complained of "unspecified GI (gastro-intestinal) symptoms." All of the four residents (see Table 5.4) who considered esophageal explanations for the target abnormality gave esophageal interpretations just after the clinical data were provided to them, and two of the four introduced the esophagus for the first time at that point. While the clinical data had much to do with residents' attempts to involve the esophagus as an anatomical localization for the target abnormality, experts either treated the GI problems as a completely independent process or interrogated the stomach area for the presence of a primary tumor from which the target abnormality (interpreted as a mass) might have metastasized or spread, e.g.:

> E3:...All right. Well, because of the GI symptoms, I'm looking below the diaphragm (an area remote from our target feature) a little bit more, and I don't see any free air. I think this is just bile and this may be stomach bubble. I can't see anything in the stomach bubble that would indicate a mass in the stomach...So, forgetting the GI symptoms for the moment and getting back to the chest x-ray...

Our best explanation for expert exclusion of the esophagus is that, by the time the clinical data was presented, experts had solidly placed the target abnormality in the lung. As our earlier discussions have suggested, residents were, perhaps, still searching for additional clues to help them build a mental representation of the patient's chest that localized the target abnormality.

V.B.4 More Case Resolution: The Odyssey Of Atelectasis

This section investigates why subjects were led to abandon atelectasis as the explanation of Film 8. The section is organized around three major types of influences involved in rejecting atelectasis. Toward this end, Table 5.6 provides a summary of subjects' investigations of the atelectasis interpretation during the analysis of the film. For each subject who ever considered atelectasis, the table includes: (1) whether or not the subject maintained atelectasis as final disposition, (2) the types of atelectasis considered (to the extent we could determine this), (3) whether or not the subject ever detected hyperexpansion in the lungs, (4) whether or not the statement

Table 5.6
Subjects' Analyses of Atelectasis (Film 8)

Subjects	Final Atelectasis	Type(s) of Atelectasis	Detect Hyperexpansion	Against Atelectasis	
				"NO CHANGE"	Other Features
Residents 1st / 2nd Year					
RA1	No	Obstruction	Yes	Yes	Saw "markings" throughout right lung
RA2	Yes	Compression	Yes	No	
RA4	No	?	No	Yes	
RA7	Yes	Obstruction, "chronic"	Yes	No	
RA9	Yes	Obstruction	Yes	No	
Residents 3rd / 4th Year					
RB2	No	?	No	?	Didn't see assoc. evidence atelectasis
RB3	No	?	No	?	Saw right heart border; Didn't see assoc. vol. loss right lung
RB6	Yes	?	Yes	Yes	Didn't see pleural reflection line
RB7	No	?	Yes	Yes	
Experts					
E1	Yes	Obstruction	Yes	No	
E2	Yes	Obstruction	Yes	No	
E3	No	"Chronic"	Yes	No	Didn't see horizontal fissure
E4	No	?	Yes	?	
E5	No-Yes*	?	Yes	?	Horizontal fissure in normal position

*Hypoplasia in right lung, specific lung component unstated.

of no change from an earlier film, as presented to the subject in the clinical data, was judged as *disconfirmatory evidence for atelectasis*, and finally, (5) other film features stated by the subject as being discrepant with an atelectatic interpretation. We next explore the factors we feel are relevant to maintaining the correct diagnosis, namely the third, fourth, and fifth columns in Table 5.6.

V.B.4.a Detection Of Lung Hyperexpansion -

Three (RA4, RB2, RB3) of the five residents who abandoned atelectasis never gave evidence of detecting hyperexpansion in the right lung and one of these (RB2) explicitly stated lack of auxiliary evidence in the film as contributing to his rejection of atelectasis.

More generally, across all subjects, residents had more difficulty detecting hyperexpansion than did the experts. All experts, in fact, detected this condition within the two-second view, as compared with only two first and second year residents and three third and fourth year residents. In their entire analyses of Film 8, seven 1st/2nd year residents and five 3rd/4th year residents eventually detected hyperexpansion.

Given the importance of hyperexpansion as a major auxiliary abnormality associated with atelectasis (either as a cause or consequence) and as a symptom of the chronic obstructive pulmonary disease which is a likely background for it, it is reasonable to presume that failure to detect hyperexpansion contributed to rejection of the atelectasis explanation among subjects who considered it (Table 5.6) and may have kept the others from considering it in the first place.

V.B.4.b Clinical Data--No Change -

The second reason some subjects rejected atelectasis as a diagnosis was the lack of a change in the radiological signs from one chest X-ray to a later one, a fact that was provided in the clinical data each subject saw. Three (RA1, RA4, RB7) of the five residents who abandoned atelectasis gave clear indication that the lack of change from an earlier film to the one we showed them (this lack of change was mentioned in the clinical data we gave them) contributed to their decisions. In addition, while another resident, RB6, did not totally reject atelectasis, his final report stated substantial doubt regarding this interpretation, explicitly because of the lack of change in the patient's chest X-rays over time. None of the three experts (E3, E4, E5) who rejected atelectasis explicitly interpreted the lack of change as disconfirmatory, although it is conceivable that this information influenced two of them (E4, E5). The other expert, E3, explicitly judged "no change" as consistent with chronic atelectasis.

Besides leading to rejection, lack of change had several kinds of effects on subjects who considered atelectasis. The one subject, RA2, who clearly advanced a compression atelectasis argument for the collapse readily assimilated "no change" to his interpretation. Two subjects (RA7, E3) with chronic atelectasis interpretations also judged the information compatible. One of these subjects, RA7, switched from obstruction to chronic collapse on the basis of the lack of change in the film. The other subject, expert E3, never really rejected atelectasis. Immediately preceding his final report, upon receiving the statement of "no change," he stated that "it is possible that he has a collapse of one of his lobes and it has remained that way." He was simply unwilling to commit himself to this interpretation without further studies--which he called for in his final report.

In general, obstruction-caused collapse arguments were, perhaps somewhat surprisingly, quite robust in the face of "no change" in the film. Of subjects clearly advancing an obstruction argument, only resident RA1 overtly cited the lack of change as reason for rejection (although RA7 switched from obstruction to "chronic collapse"). Other subjects (RA9, E1, E2) maintained their theory of a hidden (not seen on film) mediastinal mass lesion obstructing bronchial air flow and collapsing the lung. In doing so, they questioned the accuracy of the statement of "no change" or argued that the obstructing mass (even if malignant) might not change over the course of one year.

V.B.4.c Miscellaneous Incompatible Findings -

There were other film features that subjects judged discrepant with the expectations associated with collapse (see Table 5.6). While there is no general pattern to these findings, there are a few details of interest. What expert E5 saw as the right horizontal fissure (boundary between right middle and right upper lobes) in normal position largely influenced his abandonment of atelectasis for the film. What he actually saw (by his own reappraisal-- after the experiment) was a line created by the confluence of a pulmonary vessel and one border of a rib. Another subject, RB3, after raising an esophageal argument for the target abnormality based on the clinical data (GI symptoms), then claimed that he <u>could</u> see the right heart border and that this was evidence <u>against</u> atelectasis. Before that time, the subject had claimed that he could <u>not</u> see this border and had used this to support localization of the abnormality in the lung with an interpretation of atelectasis. These examples highlight the interactive nature (in both directions) of detection and the other components of the radiological diagnosis.

V.B.5 Summary Of Film 8

The analyses just presented of subjects' performance on Film 8 have shown this film to be one in which detection of the primary film abnormality was not a major source of difficulty. Rather, other components of the x-ray reading process contributed to an overall difficulty in diagnosing the film correctly. Residents demonstrated errors in localizing and interpreting the main feature in the film and proposed a broad range of interpretations associated with various anatomical components of the chest. Experts showed no difficulty in localizing the main feature correctly and were restricted to choosing between two major alternative interpretations for the feature--including the correct one.

Two of the five experts gave the correct diagnosis. A third, E5, offered an interpretation with the correct general dynamics, i.e., a hypoplastic lung area with hyperexpansion of the remaining lung areas. A fourth raised atelectasis as a strong possibility before concluding with a call for additional tests. Experts appeared to be aided in their diagnosis by:

- o a stronger association between the visual characteristics of the target abnormality and a correct medical explanation;
- o better detection of the major film features auxiliary to the primary abnormality;
- o less sensitivity to extraneous information about the patient; and
- o the triggering, almost immediately, of a general schema for the type of chest condition present, which guided and greatly constrained subsequent analysis.

For both experts and residents, the trail to correct diagnosis was fraught with hazards. Errors in abnormality detection or characterization sometimes foiled the triggering of schemas that could produce or evaluate the correct diagnosis. Compounding this was the fact that diagnostic schemas can influence what is seen (see discussion of subject RB3, last section). Also, anatomical localization constrains the range of reasonable medical explanations, and faulty localization may preclude consideration of the correct alternative. In addition, in the course of overall case resolution, a diagnostician must have an accurate medical and anatomical model of the causes and consequences of the hypothesized condition and their manifestations, in the film or in other data. For overall performance to be effective in a process involving so many interacting components, it would seem that the expert must either have greater accuracy and efficiency in the individual components themselves or a schematic organization of those components that can collectively compensate for individual component weakness, as, for example, was the case in Film 8, where a general pulmonary disease schema was triggered which constrained the localization process considerably.

V.C FILM 9: SMALL SUPRAHILAR TUMOR

V.C.1 Overview Of The Task For This Film.

The primary abnormality in this film (shown in Figure 1.2) is a small (2-3 cm) cancerous lung tumor. The position of the tumor within the lung is such that on the radiograph the tumor abuts the upper mediastinum (the central area of the chest composed of esophagus, trachea, great vessels of the heart, etc.) and intrudes on the right hilum within its upper margins.

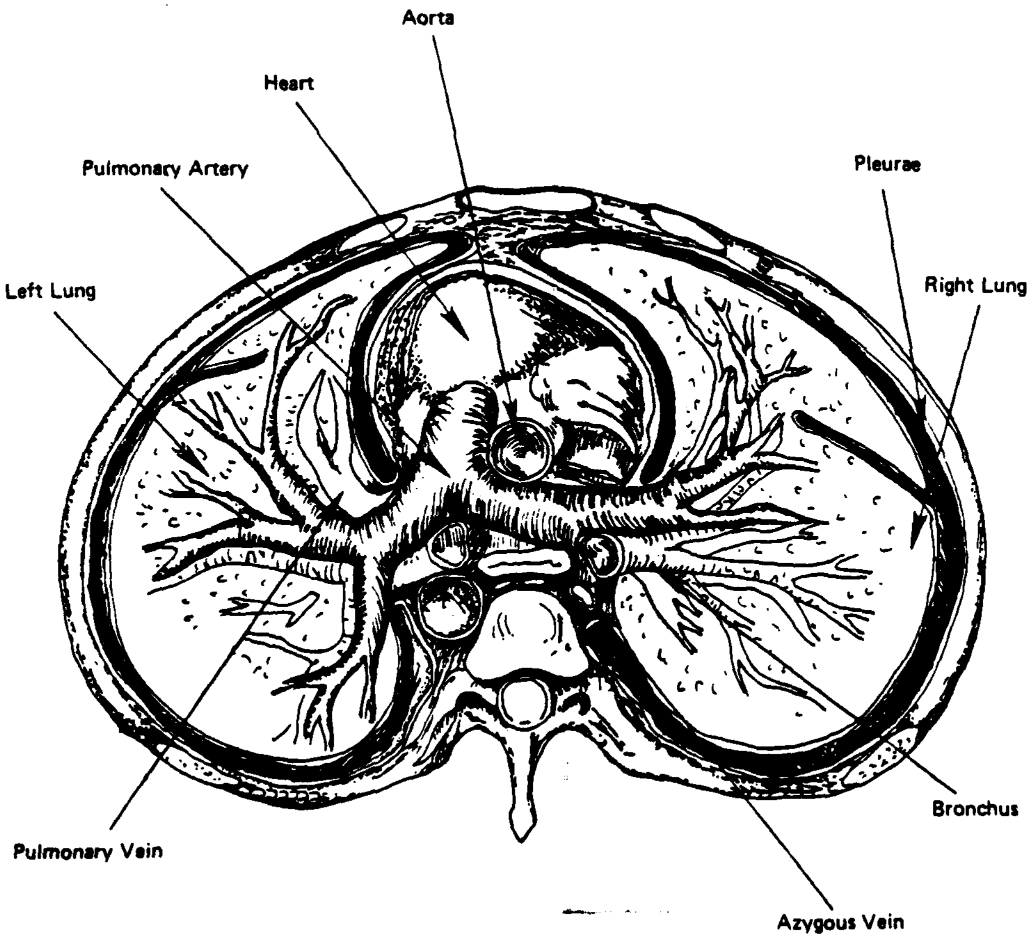

Figure 5.1 Cross-section of the chest. Note complexity in the shaded region.

The area of the chest in which the tumor is located is one in which many small anatomical components overlap. To illustrate this, Figure 5.1 shows a transverse section of the chest. The right hilum, the area of importance in Film 9, is indicated on the figure in grey. It is the boundary area in the chest between the lungs and mediastinum. The hilum is the site in which bronchi and major vessels from the heart plug into the lung, while the mediastinum is the general area between the lungs in the middle of the chest. The squashing of all the illustrated detail into a two dimensional, fuzzy x-ray picture makes the task of Film 9 very complex. However, such a small, hard-to-spot tumor is the only kind that has a modest likelihood of being treatable, so the task is important.

Film 9 differs from the previously discussed film (Film 8) in that the major abnormality is more isolated; that is, it has hardly any directly associated features within the film. The tumor is not yet large enough to change the structural properties of adjacent organs and it has not yet visibly metastasized (spread to other areas of the chest). The rest of the chest does show bilateral chronic obstructive pulmonary disease (emphysema and associated air trapping within the lungs), but this does not help in diagnosing the tumor. The clinical data we presented to subjects states only that the patient is a 65 year old woman who has complained of chest pain.

In an idealized analysis of the film, a successful reader would notice an increase in density in the right suprahilar or right superior mediastinal area. He would locate this density in the lung. A major difficulty in doing this would involve separating the target abnormality from the many anatomical components that project onto the same part of the film. Without this separation, a diagnostician might erroneously subsume the target mass into an interpretation of normal or abnormal variation of any of the anatomical structures (particularly vessels) within the region. The successful diagnostician would explain the abnormality as a mass or tumor.

V.C.2 Overview Of Subjects' Performance

In contrast to Film 8, the target abnormality in Film 9 was difficult for some subjects to detect. However, detection was only a problem among residents, especially first and second year residents. Experts detected the abnormality and did so quickly. Among subjects who detected the target abnormality, there were only two major types of competing interpretations--one involving tumor arguments (there was in fact a tumor) and another related to vascular structures. Anatomical localization issues were entwined within these alternative interpretations and, again, experts were aided by general disease schemas that were triggered early by general film features.

V.C.2.a Detection -

The target feature in Film 9 was difficult for subjects to detect; it is also difficult to decide whether a detection occurred. We established a set of scoring criteria to standardize this judgement. Creating these criteria was difficult, because most of the regional or anatomical reference points that we would use in determining a subjects' "point of view" cover large areas of the film. For example, the "right hilum" is a large area and our target feature occupies a tiny, remote edge of this area (similarly for the "mediastinum"). Hence, general references to the right hilum or mediastinum offer questionable evidence for a subject's detection of our target. Similarly, most of the specific anatomical components that "pass through" our target area also extend into large areas of the film. For example, the "aorta," which abuts the target feature in a very small portion of its entire extension, has the majority of its structure in areas remote from our target.

There were four levels of detection that we recognized:

- <u>Definite Detection (Level 1)</u>. To be credited with this level of detection, a subject had to make reference, indicating unusualness, to the <u>right supra</u> (more generally "superior") hilum or <u>right superior</u> mediastinum, or to subparts of specific anatomy localized to these regions. These were considered to be clear "hits."

- <u>Probable Detection (Level 2)</u>. This category included subjects who referred to the right hilum or vascular structure of the right hilum but never restricted their frame of reference more locally within the hilum. It is possible that these subjects saw the abnormality as including the entire right hilum, or, on the other hand, that they had detected our target but were just less precise in their speech.

- <u>Possible Detection (Level 3)</u>. Two groups of subjects were assigned to this category of detection. The first either referred to bilateral (right <u>and</u> left) hilar unusualness but never singled out the right side (which contains our target) as in any way special, or they referred to the mediastinum without ever being restricted to the specific target area. Crediting these subjects with detection of our target would be extremely generous. The second group includes subjects who raised abnormality in an anatomical component that traverses our target region but never gave indication that their judgements of abnormality specifically included the subpart <u>within</u> the target region. As it turns out, only one subject was assigned by this rule--for an unrestricted judgement of "tortuous aorta," which is a highly prevalent assessment for any film of an old patient. Other subjects who mentioned tortuous aorta were assigned levels 1 or 2 on other grounds.

- No Detection (Level 4). This assignment was used for clear lack of detection. For these subjects, there was never a pause, never a mention, never an analysis related to the target area of the film. In fact, assessments associated generally with the hila and mediastinum were normal. Subjects were assigned quite conservatively to this category.

Table 5.7
Detection of the Target Feature (Film 9)

	Levels of Detection*			
Subjects	Level 1	Level 2	Level 3	Level 4
Residents (n = 11)	(3)	(2)	(2)	(4)
1st / 2nd Year	27%	18%	18%	36%
Residents (n = 7)	(4)	(2)	(1)	
3rd / 4th Year	57%	29%	14%	
Experts (n = 5)	(5)			
	100%			

*See text for a description of levels of detection.

Table 5.7 shows the number of subjects within each experience group who were assigned to each of the detection levels just described. All experts had Level 1 detection. By the strongest criterion for a "miss" (Level 4 only), no third or fourth year residents failed to detect the target abnormality while 4 of 11 (36%) first and second year residents did. Applying the weakest criterion for a miss (anything but Level 1), would include 3 of 7 (43%) 3rd and 4th year residents and 8 of 11 (73%) first and second year residents as having missed the target abnormality. The true numbers representing subjects who failed to detect the target abnormality are probably somewhere between these extremes.

All of the experts detected the target, and two of five (40%) detected it immediately, both at Level 1. One 3rd/4th year resident (14%) detected the abnormality at Level 1 within 2 seconds, and one 1st/2nd year resident (9%) detected it at Level 2.

V.C.2.b Final Dispositions -

Table 5.8 gives the final dispositions for all subjects on the target abnormality for Film 9. Recall that the correct diagnosis is a small tumor in the right suprahilar area. The final dispositions can be used to gain an understanding of the major alternative interpretations for the key feature in the film. Each subject's highest level of detection is also given in the table so that the reader can get a sense of how closely these interpretations were tied to the target film area.

Among subjects who detected the target abnormality, there were two major types of interpretations. The first is the correct interpretation involving some kind of "tumor" (Neoplasm, Bronchogenic carcinoma, etc.). Mass/tumor arguments were concluded by 8 of the 19 (42%) subjects who conceivably detected the target feature.*9 Three of five experts (60%) gave the correct interpretation for the film. The percentage was the same among first and second year residents who detected the feature (4 of 7 or 57%), although this represents only 36% (4 of 11) of total subjects within this group. Only one of seven (14%) third and fourth year residents proposed a mass or tumor argument for the primary abnormality.

The second major interpretation for the target feature involved some variation on hilar or mediastinal vasculature and was concluded by 12 of 19 subjects (63%) who achieved detection. (Some of these subjects proposed it as an alternative to the mass/tumor interpretation.) "Vascularity" interpretations were the most common interpretations for the feature within resident groups (5 of the 7 first and second year residents who achieved detection, or 71%; 5 of 7 third and fourth year residents, also 71%), but were somewhat less prevalent among the experts (2 of 5 or 40%).

As with the previous film discussed (Film 8), it is clear from subjects' performance on Film 9 that "teasing out" the relationships among a feature and its background of complicated anatomical structure posed considerable difficulty for subjects.

In its extreme form, this problem emerged as a lack of detection that anything at all was awry, a condition confined to the least experienced subjects. Among subjects who detected the feature, we speculate there was difficulty in establishing the precise boundaries of extant anatomical structures (perhaps themselves in unusual form) so that the tumor could be seen as something additional. This problem was most acute among residents, and particularly the residents of intermediate experience. These issues will be further pursued below.

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*9 In this and later sections, unless otherwise indicated, subjects with detection at scoring levels 1-3 are treated as having detected the target abnormality. Hence, this excludes only four first and second year residents who gave absolutely no indication of detecting anything abnormal in the target region.

Table 5.8
Final Dispositions of Target Feature on Film 9

Subject	Detection Level*	Final Disposition
1st / 2nd Year Residents		
RA1	4	NO DETECTION
RA2	4	NO DETECTION
RA3	3	Tortuous *Aorta*
RA4	3	Prominent *Pulmonary Arteries*, bilaterally, from COPD
RA5	4	NO DETECTION
RA6	2	Retracted right *Hilar Vasculature* and Tortuous *Aorta*
RA7	4	NO DETECTION
RA8	2	*Adenopathy* or *Neoplasm*
RA9	1	*Mass / tumor*
RA10	1	*Mass / tumor* or Tortuous *Mediastinal Vasculature*
RA11	1	*Mass / tumor* or Normal *Hilar Vasculature*
3rd / 4th Year Residents		
RB1	2	Pectus Excavatum (a chest deformity)
RB2	1	Prominent right *Pulmonary Artery*
RB3	2	Right *Pulmonary Arterial* hypertension from COPD
RB4	3	Prominent *Hilar Vasculature*, bilaterally, from COPD
RB5	1	Normal *Azygos Vein*
RB6	1	*Mass / tumor*
RB7	1	Tortuous *Aorta* or *Aortic Aneurysm*
Experts		
E1	1	*Lymphoma* or *Bronchogenic Carcinoma* or *Metastasis*
E2	1	*Neoplasm*
E3	1	Tortuous *Aorta*
E4	1	*Mass / tumor*
E5	1	Engorged right *Pulmonary Veins*

*See text for description of levels of detection.

V.C.2.c Abnormality Feature Characterization -

Subjects used two types of feature characterizations related to the target area in Film 9. The first involved a judgement of increase in prominence or size in the region or components of anatomy. The second was a characterization of increased "mass" or "soft tissue" density.*10 Across all subjects and viewing periods, subjects were about equally likely to offer vasculature and mass/tumor interpretations in periods in which they characterized the target area by prominence/size. Specifically, of 37 subject-by-reporting-period segments in which subjects had a characterization of prominence/size, they offered a vascularity interpretation in 22 (59%) and a mass/tumor argument in 15 (41%). In reporting periods where a mass/soft tissue density characterization was advanced, mass/tumor was the overwhelming interpretation. Specifically, of 14 such periods, "mass/tumor" was offered in 10 (71%) and vascularity in only one.

These findings are not surprising since abnormality feature characterization for this film generally seemed more embedded within the anatomical localization and medical explanation processes than in Film 8. However, of possible interest were the differential responses of 3rd/4th year residents and experts to prominence/size characterizations. Out of 13 reporting periods in which the size or prominence of the target area features was mentioned by 3rd/4th year residents, mass/tumor possibilities were mentioned only once, while "vascularity" attributions were made 11 times. In experts, the situation was somewhat reversed, with mass/tumor being mentioned 7 times and "vascularity" possibilities being mentioned 4 times in a total of 9 periods for which feature size or prominence was mentioned. First and second year residents raised mass/tumor and "vascularity" interpretations in exactly equal proportions (both 7 of 15 or 47%) in response to a characterization of "prominence/size."

V.C.2.d Anatomical Localization -

Our analyses of detection of Film 9, reported earlier, showed that <u>subjects differed in the precision with which they "zeroed in" on the target feature within the film</u>. In those analyses, a subject credited with maximal detection did not necessarily engage specific components of anatomy within the target region; it was sufficient that subjects

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*10 Although they are closely related, characterization of a feature as a "mass density" and interpretation of this feature as a mass are not synonymous. "Mass density" has potentially independent meaning associated with the size and figural properties of a density (Lillington & Jamplis, 1977, p. 145). Indeed we have had subjects characterize a feature as a "mass density" and then interpret it as everything from tumor to pneumonia.

merely mentioned or referred to the region. In the Final Dispositions section above, the importance of anatomy within this region was underscored, particularly the vascular anatomy of the hilum and mediastinum. The present section extends these earlier analyses to investigate the level of detail in hilar or mediastinal vascular anatomy that subjects used during their analyses of the target abnormality.

Table 5.9
Examples of Different Types of Localization (Film 9)

Localization Type	Examples
Spatial	
	. . . Prominent right hilum or mediastinum
Anatomical: Gross	
	. . . hilar prominence . . . may be due to right hilar vasculature. . .
Anatomical: Nominal	
	. . . could be pulmonary arterial hypertension. . .
	. . . enlarged pulmonary arteries from chronic obstructive pulmonary disease. . .
Anatomical: Target	
	. . . slight density above right hilum; I think it's the azygos vein. .
	. . . Pulmonary hila themselves not enlarged. . . fullness in the right mediastinum. . . a little above the hilum. . . not part of the aorta. . . definitely separate from the aorta. . .

In this regard, anatomical localizations were classified into four categories which are given below in order of increasing anatomic specificity: (1) Spatial localizations are to two-dimensional surface areas of the film itself and are, in a sense, non-anatomical. (2) Gross anatomical localizations are to components of vascular anatomy in conglomerate without indication of which specific components are involved, e.g., "hilar vasculature." (3) Nominal anatomical localizations implicate anatomical components by name, e.g., the "pulmonary artery." (4) Finally, target anatomical localizations are nominal localizations but with explicit restriction of this anatomy to subparts within the small region of the chest containing the target abnormality. Examples of these four kinds of localizations are given in Table 5.9.

Table 5.10 shows the number of subjects within each group who achieved the increasingly specific levels of anatomical localization within their analyses of the target abnormality. Spatial localizations were all that some of our subjects generated. For example, one subject spoke of a prominent right hilum or mediastinum. Neither term refers to specific, systemic anatomy. In contrast, seventeen of nineteen subjects who conceivably detected abnormality utilized anatomic localizations. However, these localizations ranged from gross anatomy to components of specific anatomy within the target area of the film. Table 5.10 shows that this anatomic specificity was tied closely to expertise--all

Table 5.10
Most Detailed Level of Anatomical Localization for Each Subject on Film 9

Level of* Localization	Residents** 1st / 2nd Year	Residents 3rd / 4th Year	Experts
Spatial	1	1	0
Anatomical			
Gross	2	1	0
Nominal	3	1	0
Target	1	4	5

* See text for description of levels of anatomic localization.

** Excludes four 1st / 2nd Year residents with Level 4 detection.

experts mentioned specific components of anatomy within the target region, while hardly any 1st/2nd year residents did (1 of 7, 14%). The third and fourth year residents were in between. Specific vascular anatomy mentioned by advanced residents and experts included the aorta, right pulmonary artery, azygos vein, aorto-pulmonary window, right pulmonary vein, and right bracheocephalic veins.

When these findings are taken in conjunction with those from earlier analyses of abnormality feature characterization and final disposition, we can speculate that experts utilized specific anatomy largely in separating the target abnormality from (possibly other) vascular structures in the area. Third and fourth year residents, perhaps more knowledgeable of the structure of anatomy within the target region than new residents, were nonetheless largely unsuccessful in interrogating this anatomy appropriately on a radiograph. This could be due to imprecision in knowledge of the anatomical structures themselves, to limitation in knowledge of how these structures vary normally and under perturbation, or, more directly, to deficiency in mapping this anatomy onto radiographic manifestations. First and second year residents were, perhaps, more limited to recognizing gross visual properties of the film and were equally likely to respond to these with interpretations of tumor or vascularity. These process interpretations are pursued in the next section.

Table 5.11
Subjects Who Considered Various Interpretations for the Target Abnormality of Film 9

Interpretations	(n = 7)* Residents 1st / 2nd Year	(n = 7) Residents 3rd / 4th Year	(n = 5) Experts
Mass / tumor	RA8, RA9, RA10, RA11	RB2, RB3, RB6	E1, E2, E4
Vascularity (tortuous, engorged, hypertension, etc.)	RA3, RA4, RA6, RA9, RA10, RA11	RB2, TB3, RB4, RB5, RB7	E3, E4, E5
Lymphatic (adenopathy, lymphoma, etc.)	RA8		E1, E2, E4
Other		RB1 (pectus excav.)	E4 (TB)

* Excludes four 1st / 2nd Year residents who did not detect the target feature, i.e., Level 4 detection.

V.C.2.e Medical Explanation -

In this section, we investigate the interpretations subjects raised for the target abnormality during their analyses of Film 9. Table 5.11 shows the types of interpretations that were attributed to the target abnormality and the subjects within each group who proposed each interpretation even once.

The table reinforces earlier assertions that there were only two major types of interpretations for the film: vascular variations and tumor. Furthermore, Table 5.11 demonstrates that these interpretations were advanced prevalently and fairly uniformly across the subject groups; that is, the two interpretations were raised often by subjects at all levels of experience.

As discussed in the last section, first and second year residents considered these two interpretations without the benefit of anatomic detail in the area of the target abnormality that might have assisted them in making a choice. These subjects, in turn, gave both interpretations in substantial numbers as final dispositions for the feature, with an edge toward vasculature (see Table 5.8).

Third and fourth year residents considered both interpretations, utilizing anatomic detail in the troublesome area of the film (see last section). Yet, this group of subjects was ultimately the least successful in diagnosing the feature correctly (among subjects who saw it) and showed the greatest recidivism from the correct diagnosis of tumor (compare Tables 5.11 and 5.8). Final dispositions in this group were nearly uniformly "vascular," implicating deficiency in using anatomical representational knowledge in the course of the overall diagnostic process. (The first and second year residents only look better because they mentioned both major possibilities for that part of the chest rather than isolating the correct one.)

Experts raised mass and vascular interpretations (although only one, E4, raised both) and did so in the context of specific vascular anatomy within the region (again, see last section). Experts were less likely to be drawn ultimately to a vascular conclusion than either the other two groups and the expert group was the only one in which mass/tumor was the predominant final disposition (see Table 5.8). While experts were best able to separate the target shadow from variation in the patient´s vascular structure, they were not uniformly able to do so; two of five experts still concluded that the target shadow represented vessels in abnormal configuration.

V.C.2.f Overall Case Conciliation -

In this section we investigate the role of film and case context in the overall interpretation of Film 9 and in the detection of the suprahilar tumor.

Medical background. The tumor in Film 9 resides in a chest showing evidence of bilateral chronic obstructive pulmonary disease (COPD), particularly emphysema. In the medical literature, the characteristic radiological features of emphysema include the following: (1) hyperexpansion in the lungs as evidenced by increased darkness in the lung fields, (2) low diaphragms, (3) narrow and squeezed heart and mediastinum from the hyperexpanded lungs, and (4) cardiovascular changes. A common cardiovascular abnormality in emphysema is pulmonary hypertension and related enlargement of the hilar pulmonary arteries. Enlargement of the hilar pulmonary arteries, together with the other findings just outlined, is probably the "classic" description of emphysema as evidenced by presentations of this syndrome within the radiologic literature (e.g., Heitzman, 1973, p.341; Meschan, 1976, p.327; Rabin & Baron, 1980, p.389). However, enlargement of the central pulmonary arteries is a variable finding which depends on the particular biological response of the patient and the course and development of the disease; this finding need not be present.

The radiological features of emphysema and COPD are further complicated in the presence of right ventricular hypertrophy and right heart failure (cor pulmonale) which often (but not necessarily) accompany COPD and emphysema. A general effect of this complication would be to accentuate the enlargement of the hilar pulmonary arteries.

Yet a further complication arises when the lungs are not uniformly involved in the COPD. If some regions of lung are more afflicted than others, there can be shunting or diversion of blood flow away from the more diseased lung areas to areas of more healthy lung. Hence, while some hilar vessels may be enlarged, others may be diminished.

The conditions just discussed are primarily associated with the lungs and the right side of the heart (which services the lungs). Other contexts that subjects might adopt in the interpretation of Film 9 are related to the left side of the heart and systemic rather than pulmonary circulation. Chief among these are atherosclerosis and systemic hypertension ("high blood pressure"). This condition commonly causes enlargement and uncoiling of the aorta, a vessel which abuts the tumor in our film. The patient represented by Film 9 is old, probably has systemic hypertension, and does have evidence of atherosclerotic changes.

We now consider how subjects may be influenced by their knowledge of the medical models and viewpoints just discussed. It is clear from the final dispositions given by subjects (Table 5.8) that COPD played a major role in the diagnosis of Film 9. Six residents (RA4, RA6, RA11, RB2, RB3, RB4) proposed final dispositions which involved hilar vasculature; most of these implicated the pulmonary arteries and/or COPD directly. Furthermore, other subjects (RA3, RA6, perhaps RA10, RB7, E3) attributed the target abnormality to the aorta and engaged systemic hypertensive arguments in doing so.*11 We next focus on the COPD schema and the mechanisms of its influence in detection and interpretation of the tumorous mass.

V.C.2.f.i Initial Encodings -

Subjects' initial impressions of the film provide a starting point for our investigation of the role of COPD in the diagnosis of Film 9. In this regard, Table 5.12 shows the appraisals of the film components most germane to COPD which were given by subjects after their two-second viewing of the film. For each subject, the table shows which of the major, lung-related features of COPD (hyperexpansion, low diaphragms) the subject reported after two seconds, and also whether the subject gave an interpretation of COPD or emphysema for the lungs. In addition, the table gives subjects' appraisals of the heart, superior mediastinum, and hilar structures during this period.

Table 5.12 shows differences between expert and resident groups in their immediate involvement of COPD and in the breadth of implication of this condition for components of the film. Many of the residents (7 of 11 1st/2nd yr; 3 of 7 3rd/4th yr) reported none of the major lung-related features of COPD. For these subjects, initial appraisals of the heart, mediastinum, and hila were presumably made without influence of any COPD schema. Residents who reported any of the lung components of COPD varied in the number of these that they gave--from hyperexpansion only to the full set of three. Of residents who reported any lung components of COPD, only one, RB3, explicitly expressed a relationship between COPD and the other components of the chest; this was a causal relationship between COPD and a narrow heart. Although a relationship was not stated, the judgement of prominent hila by RA6 may have been influenced by COPD.

In contrast to the residents, all experts engaged elements of COPD during two seconds. The encoding of COPD was also richer; all experts except E3 engaged the full set of basic lung components of COPD. Expert use of COPD during the two-second viewing involved the chest more completely than did residents'; most judgements (except for "didn't see") of the heart, mediastinum, and hila given by experts in two seconds were given in reference to the condition of the lungs.

Upon an initial, brief presentation of the film, experts appear to have activated a schema for COPD or emphysema which carried chest-wide

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*11 In assessing how closely final dispositions given by subjects were related to the target abnormality in the film, the reader should consider the detection levels given in Table 5.8.

Table 5.12
Two Second Encodings (Film 9)

	Lungs					
	COPD / Emph	Hyperexpanded	Low Diaphragms	Heart	Mediastinum	Hila/Hilar Pulm Arteries
Residents 1st / 2nd Year						
RA1				Normal	Normal	Normal
RA2	X	X	X	Normal	Normal	Normal
RA3				Normal	Normal	Normal
RA4		X ·		Normal	Didn't See	Normal
RA5				Normal	Normal	
RA6		X		Normal	Normal	Prominent
RA7	X		X	Normal	Normal	Normal
RA8				Normal		Prominent
RA9				Normal	Normal	Normal
RA10				Normal	Normal	Normal
RA11				Normal	*Normal*	Normal
Residents 3rd / 4th Year						
RB1				Narrow	Normal	Normal
RB2				Normal	Wide	Prominent
RB3	X	X		Narrow	Normal	Normal
RB4		X	X	*Normal*	*Normal*	Didn't See
RB5				Normal	Normal	
RB6	X	X	X	Normal	Normal	Normal
RB7		X	X	Upper Limits	Normal	Normal
Experts						
E1	X	X	X	Narrow	Narrow	Didn't See
E2	X	X	X		Didn't See	Didn't See
E3		X	X	Narrow	Wide	Didn't See
E4	X	X	X	Normal ("for age")	Narrow	Diminished
E5	X	X	X	Narrow	Normal	Prominent

implications. Interrogation of schema-relevant components (e.g., the state of the heart) was schema-driven once the schema was engaged and served earlier to activate the schema. Novices, in contrast, appear not to have immediately engaged a chest-wide schema for the film; immediate interrogation of film components was piecemeal for them.

Although residents show little evidence of having engaged a rich COPD schema within two seconds, their final dispositions, as discussed earlier, suggest that these subjects eventually did engage such a schema and that this schema carried major implications for hilar vasculature, particularly the hilar pulmonary arteries. Since only experts expressed a relationship between COPD and hilar vasculature during two seconds, we must look at their data for clues to what happened eventually among the residents.

Within two seconds expert E4 saw the hilar areas as <u>less</u> than usually prominent and marked this as completely consistent with COPD:

> E4...(lungs) definitely hyperaerated. Hila less than usually prominent which is all part of emphysema. Mediastinum narrow and squeezed upon by the hyperaerated lung fields. Definite flattening of the diaphragm. I would put this as moderate to far advanced emphysema...

Such a view would predispose a subject against later interpreting the hilar tumor as hilar vascular structure.

In contrast, expert E5 adopted a framework for COPD that included prominent hilar structure, particularly in the pulmonary arteries:

> E5...The lungs were overinflated. This usually means chronic obstructive airways disease or air trapping in a symmetrical and diffuse manner. However, occasionally, some patients are able to take a deep breath in and limit this finding. The hilar structures appear normal relative to each other, but they did appear to be prominent. This prominence is consistent with a degree of increasing pulmonary arterial pressure that is occurring symmetrically and bilaterally...

This view could predispose a subject toward subsuming the added film density caused by our tumor within an interpretation of abnormal vascular structure.

The other three experts (E1, E2, E3) were non-committal in the two-second viewing regarding the hilar structures and we have recorded them in Table 5.12 as not having seen the hila. Their statements were ambiguous; we cannot be sure whether they did not have time to look at the hila or whether the hila were appreciated in some way as non-prominent:

> E1...I didn't see the hila. I didn't see either hilus. Again

> it's a voluminous lung...
>
> E2...The hila, I don't think impressed me. The lung fields were very hyperlucent...
>
> E3...I thought the lung fields were rather hyperlucent. I didn't appreciate the hila very well...

We do know that none of these subjects ever interpreted the target density in Film 9 as hilar vasculature and that, even though they engaged a COPD schema early, it did not constrain them toward a strong commitment to enlarged hilar structure.

As we have noted previously, the model of emphysema which includes enlarged pulmonary arteries is probably the most common version and the predominant version in medical descriptions of the disease. In this sense it is the classic picture of the condition. However, emphysema comes in numerous taxonomic variants, all of which are sensitive to extenuating factors of the host. Other investigations of medical diagnosis have shown diagnosticians such as our residents to be more constrained to the "textbook" or classic versions of diseases than the more experienced diagnosticians are and to be less able to adjust their ideal schemas to context (Feltovich, 1981; Johnson, Barreto, Hassebrock, Moller, Prietula, Feltovich, & Swanson, in press). These same investigators have shown that experts are better able to "extract" themselves from classical interpretations when it is appropriate to do so. In our current studies, this flexibility is again demonstrated by expert E5, who immediately discarded his theory of enlarged pulmonary arteries upon closer examination of the film during the first full film analysis:

> E5...The hilar structures are somewhat surprisingly small for the degree of overinflation of the lungs. This is a rather unusual finding as I've indicated because in this degree of hyperinflation, we would expect much larger main pulmonary arteries...

In this instance, we might presume that the mechanism of expert flexibility involves the ability to build and directly access a detailed mental representation of anatomical structure, as earlier results presented for Film 9 have suggested.

That residents were handicapped on Film 9 by an overly restrictive COPD schema is further indicated by Table 5.13. For all subjects who engaged even the rudiments of a COPD schema during two seconds, the table shows the final dispositions of these subjects for the target abnormality. Half of the residents who engaged COPD early ended up with an interpretation of the target feature as hilar vasculature; only one expert concluded hilar vasculature and his final disposition excluded pulmonary arteries.

Table 5.13
Final Dispositions for Target Abnormality of Subjects Who had an Early "Model" of COPD

Subjects with COPD in Two Seconds	Final Dispositions for Target Feature			
	No Vasculature	COPD: Vasculature		
	COPD Only	Hilar Vasculature	Mediast. Vasculature	COPD: Tumor
Residents				
1st / 2nd Year				
RA2	X			
RA4		X		
RA6		X		
RA10	X			
Residents				
3rd / 4th Year				
RB3		X		
RB4		X		
RB6				
RB7			X	X
Experts				
E1				
E2				X
E3				X
E4			X	
E5		X		X

V.C.2.f.ii The COPD Schema And Later Processing -

This section of the report further elaborates the relationship between the COPD schema and eventual diagnoses of hilar vasculature versus tumor. It was shown in the last section that residents varied considerably in the extent to which they had engaged a COPD schema during the first two-second viewing of the film. Yet, the schema eventually played a significant role in shaping their final dispositions. This section further investigates the relationship, in subjects' diagnoses, between COPD and the interpretation of the suprahilar abnormality as hilar vasculature or, correctly, as tumor.

If we use as a criterion that a subject, at some time during analysis of the film, raised at least the primary lung components of COPD (i.e., COPD, emphysema, or hyperexpansion of the lungs), then all but one subject, RA3, can be shown ultimately to have engaged at least the rudiments of a COPD model. As a guide to determining the relationships of interest in this section, we can investigate the sequential ordering in each subject's protocol of statements related to COPD and interpretations of the target abnormality as tumor or hilar vasculature.

In this regard, Table 5.14 lists all subjects who ever interpreted our target abnormality as hilar vasculature or as mass/tumor.*12 The table also roughly captures the sequencing for each subject of these interpretations with those of COPD. For these three interpretation categories, the table shows the segment of film analysis in which the subject <u>first</u> proposed the interpretation. For COPD, this means the first proposal of COPD, emphysema, or hyperexpansion in the lungs. In addition, the asterisks in the table show the interpretations concluded as final dispositions by each subject.

Table 5.14 shows three main patterns involving interpretations of hilar vascularity and tumor. There were subjects who considered hilar vascular interpretations only, subjects who considered tumor only, and subjects who considered both. Each pattern will be addressed individually.

<u>Hilar vasculature only</u>. Four subjects (RA4, RA6, RB4, E5) raised only hilar vasculature as an explanation for the target abnormality and all four maintained vascular interpretations as final dispositions. Of this group, the three residents raised a vascular interpretation for the first time during the first free-view/formal-report period and had proposed COPD during the initial two-second view. Two of them (RA4, RB4) linked vascular interpretations directly to COPD, e.g.:

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*12 Again, the detection levels shown in Table 5.10 can serve as a guide to how closely these interpretations were tied to the target area of the film.

Table 5.14
Sequential Order of Selected Interpretations (Film 9)

	Interpretations		
Subjects	COPD	Target As Hilar Vasculature	Target As Tumor
Residents 1st / 2nd Year			
RA4	1	2*	
RA6	1	2*	
RA8	3		2*
RA9	2	2	3*
RA10	2		3*
RA11	2	2*	2*
Residents 3rd / 4th Year			
RB2	2	2*	2
RB3	1	2*	3
RB4	1	2*	
RB6	1		2*
Experts			
E1	1		2*
E2	1		2*
E4	1	2	2*
E5	1	1*	

Note. 1 = two-second viewing, 2 = first full analysis or first formal report, 3 = second full analysis or second formal report.

* Indicates inclusion within subject's final disposition for the target feature in the film.

> RA4...The mediastinum is thought to be slightly enlarged bilaterally. It's probably due to the prominence of the pulmonary arteries, secondary to the patient's chronic obstructive pulmonary disease...

These subjects seem ultimately to have engaged the classic disease schema for COPD, which then mediated interpretation of the target abnormality (to the extent that the target was detected at all as a separate entity).

The expert, E5, who proposed only vascular interpretations was discussed in the last section of this report. He proposed an interpretation involving the pulmonary arteries during the two second viewing, rejected this during the first full film analysis, but ended up with an interpretation involving bracheocephalic veins. The relationship between these interpretations and COPD is complex and will be addressed separately below.

Tumor only. Five subjects (RA8, RA10, RB6, E1, E2) raised only tumor interpretations for the target abnormality in the film. Three of these subjects (RB6, E1, E2) generated tumor for the first time in first free-view/formal-report period after having engaged a relatively rich schema for COPD during the initial two-second view. Perhaps significantly, experts E1 and E2 were non-committal regarding hilar vascular structures during the two-second viewing.

Despite early invocation of the COPD schema, the tumor-only subjects were not led, as were the last group of subjects discussed above, to hilar vascular interpretations. Among possible explanations for this contrast might be: (1) that the disease schema(s) for COPD engaged by the current subjects was less constraining in its expectations regarding the hila than the model engaged by other subjects, (2) that the current subjects were better able directly to relate components of their anatomy representation for the patient to features within the film, or (3) there was a more flexible interaction between schema-driven expectations and the more direct perceptions of vascular structures.

Subject RA10 was similar (tumor after COPD) except that he did not raise COPD until after the two-second initial view and did not propose an interpretation of tumor until the end of his film analysis, during the second formal report. However, all assessments of COPD by the subject were of at most "moderate" severity and there was never a clear commitment to the presence of this condition in the film. All references to the target film abnormality before the second formal report were to the aorta, and mediastinal vasculature was included as an alternative to tumor in the final disposition (Table 5.8). The diagnosis by this subject seemed more dominated by a systemic (left heart) point of view than by pulmonary models.

Finally, subject RA8 detected nothing of COPD until his final film analysis, after he had already made an interpretation of tumor. Hence, possibly confounding influences of a COPD schema had less opportunity to exert themselves.

Hilar vasculature and tumor. Five subjects (RA9, RA11, RB2, RB3, E4) proposed both hilar vasculature and tumor interpretations for the target abnormality at some point during their diagnosis of Film 9. Of the four residents within the group, two had a final disposition including only hilar vasculature. One, RA11, gave a final disposition including both tumor and hilar vasculature, and the remaining resident, RA9, concluded only tumor. The final disposition of expert E4 was tumor.

Table 5.14 shows that two residents (RA11, RB2) generated interpretations of COPD, hilar vasculature, and tumor, for the first time, all within the same period. Specifically, these were all within the first full analysis of the film. Resident RB2 proposed vascularity and mass together as interpretations and immediately rejected tumor in favor of pulmonary arteries:

> RB2...Uh, the mediastinum, what I thought was some prominence above the right hilum, I think is maybe just a prominent pulmonary artery rather than a mass, and the heart size and configuration look all right...

This was the only interpretation as a possible mass within this subject's entire diagnosis of the film, and his final disposition was as prominent pulmonary artery and COPD.

The protocol of the other resident, RA11, from the first full analysis is given in total as Figure 5.2 because it nicely illustrates the interaction between COPD and interpretations of tumor and vascularity. The protocol shows that "mass," among the three interpretations, was raised first (first underlined segment--Figure 5.2). The subject saw the right hilum as prominent but distinguished in the hilum between its inferior and superior (our target region) aspects. The former he interpreted as vasculature, the latter as possibly a mass. Later in the protocol, the subject raised the interpretation of COPD for the first time (second underlined segment). Then, in the formal report which followed, the entire "prominent" right hilum was interpreted as vascular structure, and this interpretation was set within a "picture" of COPD. This general stance was maintained through the second analysis and report, although a mass possibility was again raised, but as a less likely possibility.

Resident RA11 was like resident RA8, discussed in the last section, in that he, in a sense, benefited from having developed his COPD schema late. In general, the earlier that residents engaged COPD, the less likely they were to conclude tumor over hilar vasculature. For example, of the five residents recorded in Table 5.14 as having raised COPD within the two-second viewing, four had final dispositions involving hilar vasculature and only one concluded mass.

Yet, the two remaining residents to be discussed in this section provide examples of subjects who were able at least partially to break the influence of COPD in the interpretation of the target abnormality. Both of these subjects engaged a COPD schema and generated a strong COPD-related vascular interpretation before generating tumor in the last full analysis period (after seeing clinical data). For both of these subjects, it was the clinical history of "chest pain" that provided the external impetus. For subject RB3, the COPD schema and its constraints were too dominant, and the consideration of tumor almost rhetorical:

First Full Film Analysis

Okay - well there may be some asymmetry of the breast shadow, but I don't know if that's — it's probably within normal limits, certainly both are present. Um, the heart is not enlarged although it may have a left ventricular contour. Uh, there's calcification in the aortic arch and probably some unfolding of the aorta. The mediastinum is not widened. *There is some prominence of the right hilar shadow – at least the inferior part of the hilar shadow I believe is all vascular and there is a suggestion of a mass in the right suprahilar region.* Uh, again there's a sort of a — well, there's a diffuse increase in the interstitial markings in both lungs — perhaps a little more accented at the basis, but not much difference between the bases and the apices. Um, no pleural effusions,– *there's hyperinflation of the lungs with flattening and scalloping of the diaphragm and therefore I'd raise the question of emphysema in this patient.* I don't see anything definite that looks like bullae or blebs although there is a suggestion of some small rounded densities on the left lower lung zone which may represent small blebs. I don't see any significant pleural reaction at the lung apices. Uh, the visible bones are markedly osteopenic — uh, I don't see any fractures — no pseudo-fractures in the clavicles. It's probably secondary to post-menopausal osteoporosis — OK.

First Formal Report

So the report — hyperinflation of the lungs and increased interstitial markings with prominence of the right hilar shadow which may be all vascular. These findings suggest chronic obstructive pulmonary disease. The heart is not enlarged and there's diffuse osteopenia.

Figure 5.2 Protocol from subject RA11, Film 9.

> RB3...Okay—some chest pain. Looking at it - got chest pain - so I'm looking then - I'm saying to myself "did I miss?"--you know the right hilum is awfully prominent there but I think that's all pulmonary artery from her COPD. If she has a carcinoma in that area, it's too bad because I'm not going to be able to see it...

The representation of COPD constructed by the subject for the film included cor pulmonale (right heart failure) which carries even greater commitment to pulmonary arterial hypertension than does COPD without this complication; the subject's final disposition was as pulmonary arteries. For the other subject, RA9, the clinical information prompted a further looking and an ultimate disposition of tumor:

> RA9...A 65-year old female with some chest pain--well the fact that she's 65 goes along with the bones, so the osteoporosis is OK. And some chest pain doesn't help me. I didn't see any etiology for her chest pain -- unless she does have a right hilar mass. And so, I guess I'll have to look at that again and try to commit myself one way or another at the right hilum...

Two other subjects, both experts (E1, E4), used the clinical evidence of "chest pain" in support of interpretations of tumor. However, for these subjects tumor was a pre-existing hypothesis and "pain" functioned not as a trigger, but as an item of confirmatory evidence in overall resolution of the case.

Expert E4 considered both tumor and vascular interpretations. While he initially encoded the hila as diminished, he generated both tumor and vascular interpretations during the first full analysis and report -- the vascular interpretation was not general to the right hilus, but was precisely localized to its superior aspect. Again, it was external context that tipped the balance in favor of tumor. In addition to "pain," the subject drew upon the clinical coincidence of emphysema and lung cancer, and their common etiology:

> E4...Nothing I didn't expect. Chest pain - there are many causes of chest pain. When you see this in an individual, particularly an emphysematous individual who smokes and is therefore at risk for lung cancer, I would be doubly cautious about that right hilus. Other than that, I don't think we have new information here...

The subjects discussed in this section emphasize both the potentially detrimental and the potentially beneficial effects of context in the perceptual diagnostic process. In the course of diagnosis, emergent conceptualizations direct and predispose other components of the diagnostic process. Yet, the successful diagnostician is opportunistic; countervailing influences are constantly available (at least potentially) from (a) the results of further looking, (b) a rich set of alternative schemas, (c) precise schema-embedded tests that verify triggered schemas by looking for what <u>should</u> <u>be</u> <u>there</u> if the schema is correctly triggered, (d) a precise mental representation of anatomy that supports accurate schema testing, and (e) rich <u>clinical</u> (medical) knowledge that mediates conflicts. The effective diagnostic system is a system of checks and balances.

V.C.3 Summary Of Film 9

Multiple sources of benefit and detriment. Film 9 is one in which the target abnormality presents a "weak signal," to use signal detection terms. The tumor presents a very small area of density that is not particularly characteristic in its shape and is not even distinctive in its degree of opacity from its surrounding of anatomical structures. Furthermore, there are no auxiliary pathways to the correct diagnosis (in contrast to Film 8, say, where atelectasis could potentially be suggested by the general state of the lungs).

Subjects' performance confirmed the lack of saliency in the target abnormality. Depending on our criteria for detection, between 22% (Level 4 detection only) and 61% (anything but Level 1) of residents failed to see the target feature as a distinct entity.

In general, detection improved with the degree of experience of the subjects. All experts detected the abnormality by our most stringent criteria.

In contrast to the target abnormality, other film features were easily detectable, and highly influential in the diagnosis of the film. The lungs were big and dark, and the diaphragms down; almost no subjects failed to establish COPD and emphysema in the chest. These schemas include strong expectations for the state of chest region occupied by target abnormality. Since the target was indistinct, much of the analysis of it was mediated by subjects' expectations.

In the results of the previous film, Film 8, it was shown how a disease schema, an integrated set of radiological expectations, could benefit the analysis of a specific film feature by setting appropriate constraints. Film 9 has demonstrated the opposite effect, where schema-driven expectations largely led to error. Many subjects were influenced to interpret the target abnormality as the typical vascular pattern of emphysema.

However, some subjects were able appropriately to avoid or circumvent the potentially deleterious effects of schema-driven embellishments. In this regard, several kinds of mechanisms were seen to serve as checks on the diagnostic process.

One source that countervails conceptually driven embellishment is a detailed and rich mental representation of the particular patient's anatomical structure. Subjects differed in the detail of anatomy they constructed for the target region of the film, and this detail increased with their experience. Anatomic detail serves as a check-mechanism on the expectations associated with hypothesized conditions. For subjects under a COPD schema who expected the pulmonary arteries to be prominent and who were only capable of seeing the hilar structures as an undifferentiated tangle, the density corresponding to the tumor simply reinforced expectations. In contrast, subjects able to discriminate among specific anatomical components could test their model of COPD more critically.

Yet the mechanisms of proficient diagnosis are not this simple and unidimensional. For example, third and fourth year residents showed evidence of constructing a detailed representation of anatomy within the target region but were nearly uniformly unable to distinguish these anatomical components from the tumor. A partial explanation lies in the variability of anatomic structures. There is no <u>single</u> chest anatomy for a diagnostician to master. The anatomy of the chest is context sensitive; the chest anatomy of a healthy 24 year old is not the chest anatomy of a 70 year old, emphysematous hypertensive individual. Hence, the construction of a representation of anatomy for any particular patient will be more accurate the more it takes account of the entire condition of the patient. Conversely, a diagnostician's emergent conception of this context will largely guide the representation of expected anatomy that he constructs. For the "tumor" to be seen as expected anatomy plus "something extra," the representation of expected anatomy must be contextually appropriate. Hence, context and the representation of context by subjects might be expected to greatly influence their detection of the tumor.

One element of context is the disease condition of the patient. The performance of subjects suggests that they differed in the disease schemas which they attempted to fit to the condition of the patient. Resident schemas of COPD were classic versions of this condition, with "textbook" specifications of the radiologic appearance of the pulmonary arteries. These expectations either directed interpretation of the pulmonary arteries as enlarged, or, alternatively, provided a receptiveness to this interpretation when it was made on other grounds. Expert COPD schemas were richer, specifying implications over a broader range of the chest, but more tolerant regarding the state of the pulmonary arteries and hila. We can speculate at least two possible sources of this increased tolerance in the expert. One might be that the features of a disease and their expected radiologic manifestations represented within the disease schema are better "tuned" to the clinically occurring variability of the feature; the range of allowable values in a schema "slot" are (in the present instance of pulmonary arteries) appropriately broad. A second might be expert knowledge of more alternative subschemas of chronic obstructive pulmonary disease, defined by differences in severity, time course, or physiologic type. If the features specified under one disease variant are violated by evidence, other variants are then available to be fit to this evidence (see example later in this section; also Feltovich, 1981, Johnson et. al, in press). By either of these mechanisms, a diagnostician would have less need to "force" an interpretation along schema-specified lines, but would be more receptive to countervailing evidence from other sources, e.g. the developing representation of the patient's anatomy.

Another attenuating influence on interpretive conceptions that develop during film diagnosis involves the <u>breadth of available context</u> utilized by the diagnostician. We have seen that a wide ranging representation of the patient's condition can serve in testing hypotheses and generating new ones. An example from Film 9 was some subjects' use of the clinical history of "chest pain." For subjects who were already considering tumor the information served in support. For some other subjects strongly under the influence of COPD and vascular interpretations for the target abnormality, the realization that these conditions alone are not likely to cause chest pain served as an opportunity to break the COPD "set" and triggered the consideration of tumor. Another example involved a rich relationship in the diagnostician's medical knowledge between two diseases and their common etiology. This enabled the subject, expert E4, to integrate his two major competing hypotheses, COPD and tumor, within a single interpretive framework, mediated by a common etiological factor in both conditions--a history of smoking. Through this richness of representation the two conditions were seen not as alternatives, where one was to be chosen over the other, but as the same condition at a higher level of integration.

A final set of examples. In closing this section, we will return to a segment of protocol from expert E5 because it demonstrates some of the mechanisms that have been discussed above and raises some others (this segment was also discussed in Chapter I). The segment to be discussed is from the subject's first full analysis of Film 9, which follows the two-second viewing of the film. The protocol presented is intact and in order of occurrence, but segmented for annotation.

Recall that this subject, during the two-second viewing, had engaged a rich schema for COPD, with broad implications of this condition for the components of the chest, including a small heart (see Table 5.12). The schema of COPD was the classic version and the pulmonary arteries were assessed or seen as prominent. This was also a subject who detected the target abnormality within two seconds and raised an interpretation involving pulmonary arteries.

In the first film analysis, after substantiating the emphysema in the lungs, the subject analyzed the pulmonary arteries and found his initial preconceptions in error. Direct perception of anatomical structure in detail prompted a change in hypothesis:

> ..The hilar structures are somewhat surprisingly small for the degree of overinflation in the lungs. This is an unusual finding as I've indicated because with the degree of overinflation one would expect much larger pulmonary arteries...

The change in hypothesis was to a different variant of COPD, one consistent with a degree of left heart failure and corresponding pulmonary venous hypertension:

> He seems to have pulmonary venous hypertension which is accounting for that area in the right supra hilar region that I was uneasy about earlier on...

This conception is, in turn, checked by a physiologic "critic," which detects inconsistency with constraints of physiology having to do with pulmonary venous hypertension and heart size:

> I'm trying to tie up this venous hypertension with the relatively non-enlarged heart...

The result of criticism is a switch to yet another variant of COPD, the version with non-uniform involvement of the lungs (see Section V.C.1):

> OK, the other explanation is that the diversion of blood flow to the upper pulmonary veins may be secondary to chronic lung parenchymal disease in the lower lobes--and that could be an alternative explanation. In that, he does not really have pulmonary venous hypertension, but he has diversion due to destructive lung disease in the lower lobes...

The subject maintained this COPD theory through the first formal report on the film (given below) and all subsequent film analyses:

> ..There is pulmonary venous diversion to the upper lobes. This is in keeping with chronic obstructive airways disease which is most evident at both (lung) bases, but present, though to a lesser extent, in both upper lobes. Sorry, present to a lesser extent in the remainder of the lungs bilaterally...

This subject demonstrates both the potentially detrimental and beneficial effects of schema-driven embellishment. Schema-driven expectations, in some instances, provide acuity in testing hypotheses against the data and, in others, prejudice the testing process. The subject also demonstrates some of the kinds of mechanisms available to temper emergent conceptions, including astute direct perception of anatomy, a rich substrate of medical and biological constraints for criticism, and a rich store of alternative interpretive frameworks. Yet, in this instance, the interpretive schema, despite all of its adjustments, was still basically detrimental to arriving at the appropriate interpretation of the film.

In this section we have proposed perceptual diagnosis as a complex, interactive process of constructing an interpretive theory of a particular patient, the patient represented by the film. The process takes cues from the film (and other available sources of information), embellishes these from a rich medical knowledge base, criticizes and transforms these embellishments, and projects back onto the available evidence. This process recurs as new information is accumulated either through additional looking or through the internal products of theory building.

V.D FILM 2: A FILM WITH MULTIPLE LESIONS

The two films that have been discussed so far show different structural characteristics. Film 9, the film just presented, involved a single target abnormality, with no structurally related abnormalities. The prior film, Film 8, also contained a single but more salient abnormality. However, in contrast to Film 9, Film 8 contained abnormal manifestations throughout the chest that were structurally related to the target abnormality. The film to be discussed in this section, Film 2, represents yet a different type of film. Film 2 contains multiple features of abnormality, most of which are readily apparent, and there is no predominant main abnormality. The etiological relationships among these abnormalities are the primary problems to be resolved in diagnosis of the film. Analyses at the levels of detail of the previous film discussions above will not be presented for Film 2. Rather, we will concentrate on results that relate to the multi-abnormality issue.

V.D.1 Overview Of The Task For This Film

The film (shown in Figure 5.3) is the case of a 71 year old female who had had an aortic valve replacement and who, at the time of the x-ray, complained of chest pain and shortness of breath, although she had no fever. This information was supplied to our subjects in the clinical history phase of the task.

The chest x-ray for the case contains numerous features of abnormality. The lung fields contain three ill-defined round densities: one in the right middle lung, another larger density opposite the first in the left middle lung, and a third density in the left lower chest, overlying the apex of the heart. In addition to these densities, the mediastinum is wide, and the heart is moderately enlarged with a metallic density which represents the prosthetic aortic valve. There is pleural effusion in the right lower side of the chest. The bone of the right upper arm (humerus) shows degenerative changes and there is an absent right breast. Suture material from previous thoracic surgery can be seen in the midline of the chest and two electrocardiogram leads appear as well-demarcated, round metallic densities on each side of the chest.

Most of these features are easily detected, but interpretation of the features is variable as is the apportioning of combinations of features to specific etiologies. Several of the abnormal features, the somewhat enlarged heart, the lung densities, and the pleural reaction, can plausibly be attributed, more or less directly, to congestive heart failure. The evidence of surgery (without knowledge of its recency) supports an alternative line of reasoning in which some of the chest features are seen as post-surgical reactions (e.g., mediastinal bleeding as an interpretation for the wide mediastinum). The post-surgical and heart failure arguments are not necessarily distinct, as heart failure can occur as a surgical complication. Surgical arguments are reinforced

Figure 5.3 Film 2.

by the (coincidental) presence of the EKG monitor leads, although the patient's chest surgery for aortic valve replacement had occurred three years prior to the x-ray. Finally, the right humeral (arm bone) lesions and the absent right breast support yet a third line of reasoning involving cancer and metastasis (spread) to other components of the chest. The strong cues to the various conditions discussed above make part of the diagnostician's problem one of determining which of the collection of abnormalities within the chest are to be subsumed under the alternative explanations.

The medical record of the patient represented by the x-ray substantiates that the patient did have metastatic involvement in the chest and had had a prior breast removal for breast cancer. Further, at least some of the three lung densities were due not to tumor but to areas of pulmonary infarction. However, the precise etiologies for all of the abnormalities could not be determined.

V.D.2 Final Dispositions

The analyses of the film given by subjects before they receive clinical data on the patient substantiate the considerable ambiguity in the film itself. This can be demonstrated by the dispositions for the set of abnormalities in the film that subjects held prior to receiving clinical data. For this purpose, Table 5.15 shows the main explanations subjects advanced for these abnormalities before they received clinical data.

Table 5.15 records subjects' final dispositions for film abnormalities within five categories.*13 Cancer, congestive heart failure, and surgery have been discussed above. "Description only" implies that the subject only described the abnormality (e.g. "wide mediastinum) without offering an interpretation. The "other" category contains numerous interpretations, which vary with abnormalities and are not subsumed within the three main medical arguments. It should be noted that some of the interpretations listed under "other" may be identical to interpretations listed at other times under another category. They are listed under one of the more specific categories when the subject implied a causal or subsidiary relationship of the otherwise uncategorizable argument to an argument of cancer, heart failure, or surgery. For example, edema, when applied to one of the lung densities, is listed under "failure" when the subject indicates the edema is the consequence of failure; it would be listed under "other" when no such relation to one of the tabulated arguments is given. In this sense, we have attempted in the table to capture major lines of interpretation for the film at a level of abstraction above first-order interpretations (e.g. edema, infiltrate, hemorrhage, etc.), many of which are shared by the candidate medical explanations.

It can be seen from Table 5.15 that there is no general consensus explanation for the film, either across subjects or within groups of subjects at any level of experience. At least one abnormality is attributed to heart failure by nine subjects (39%). When "failure" is advanced by subjects as an explanation, it is applied to from two to six

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*13 The possible interdependence (for example, disjunction, conjunction, hierarchical) among arguments applied to abnormalities, were too complicated to capture in the Table 5.15. The table listings should be interpreted simply as implying that the subject applied the argument as final disposition in some way to the abnormality listed.

Table 5.15
Final Preclinical Arguments Used to Account for Features – Film 2

	Mediastinum	Hila	Heart	Lungs (General)	Lung Densities	Pleura	Breast	Arm
1st / 2nd Year Residents								
RA1	O		D		C	O	D	C,O
RA2		F,S,O	F	F,S,O	F,S,O	O		C
RA3	F,C,O	F,C,O	F	F,C,O	F,C,O	F,C,O		C
RA4	D,O		D		D	O	D	D
RA5		C	D	C,O	C	O		C
RA6	C	C			C		C	C,O
RA7		F,C	F	F	C	F	C	C
RA8		F,C	F	F	F,C,O	O	D	C
RA9	F,C	F,C	F		F,C,O	F,O		C
RA10	D	D	D	D	D	D		D
RA11	O	O	D		O	O		C
3rd/4th Year Residents								
RB1	F,C	F,C	F	F,C	F,C	F,C	D	C
RB2	F	F	F		F,C	F		C
RB3	C,O		D		C	D	C	C
RB4		C,O			C,O	C,O		C
RB5		C	D	C,O	C,O		C	C
RB6		O	D	O	O	O		D
RB7	C	F	F		C			C
Experts								
E1	O,S			O	O,S	O		C
E2	C	C,F	F		C,F	F,O		C
E3	O		D		O	S,O	D	D
E4	C	C	D		C,O	O		C
E5	O	S	S		O	O		C

Legend:
C = Cancer
D = Description Only
O = Other Unintegrated Interpretations
F = Failure
S = Surgery

(mean = 4.4) features of the chest. At the larger end of this range, a subject is advancing a "picture of failure" for the chest. Because of the degenerative lesions in the right arm, nearly all subjects (19 of 23, 83%) included cancer arguments within their final dispositions for the film. Excluding the arm and absent breast, cancer was still concluded by fifteen (65%) subjects for at least one abnormality within the chest itself. When cancer was used by subjects to explain chest abnormalities, it was applied to from one to five (mean = 2.7) chest abnormalities, exclusive of the arm and breast. Again, at the upper limits of this range, a subject is proposing a pervasive "picture of cancer" for the chest. Cancer and failure explanations were offered for chest abnormalities by residents and by experts (Cancer: experts 40%, residents 72%. Failure: experts 20%, residents 44%). Interpretations of abnormalities as resulting from the patient's thoracic surgery were given by only one resident but by three of the five experts.

In addition to interpretations as areas of cancer or as lesions resulting from heart failure, the three lung densities in the film were given a wide range of interpretations including pneumonia, hemorrhage, sarcoidosis, aneurysm, edema, and embolism/infarction.*14 Importantly, embolism/infarction, the "correct" diagnosis for at least some of the lung densities, was an infrequent interpretation for these abnormalities prior to seeing the clinical data for the patient. Only two residents (11%) and two experts (40%) concluded this interpretation for a lung density prior to clinical data being presented, and only six subjects even raised this interpretation during pre-clinical analyses of the film.*15 As will be seen, this is in distinct contrast to the prevalence of this interpretation after subjects are given clinical history about the patient, suggesting the importance of external context in resolving the radiologic ambiguity of the film.

V.D.3 Initial Encodings

The ambiguity in the film is reflected in the initial impressions generated by subjects during the two-second viewing period. In this regard, Table 5.16 shows the major explanations (congestive heart failure and cancer) that were offered by subjects for at least one abnormality (excluding arm and breast) during the two-second period. These explanations were used to cover a range of observations, from isolated, single abnormalities to a broad-ranging conception of the

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*14 Pulmonary embolism and pulmonary infarction are closely related causally and often interchanged by subjects. We treat the terms as the same in this section.

*15 Subjects with interpretations of heart failure causing embolism/infarction in the lung densities were, of course, credited with an embolism/infarction interpretation for the densities.

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entire chest.

It can be seen from Table 5.16 that many subjects (52%) developed impressions of cancer for the chest early in the viewing of the film. Early conceptions of heart failure were somewhat less common (22% of subjects) and were confined to residents; no expert activated an interpretation involving heart failure as a result of the two-second viewing.

Taken in conjunction with those concerning final dispositions (Table 5.15), the initial encoding results suggest that congestive heart failure played a less important role in the analyses of Film 2 for experts than it did for residents. It should be noted that when subjects were asked, on a questionnaire, to list the abnormalities they see most often in chest films, 15 of 18 residents (83%) listed heart failure or directly related conditions among their three most frequent abnormalities. Only one of five experts listed this condition. This is another example of how their working experience may affect resident's interpretations of films.

Initial interpretations of the film which were not associated with the major arguments tabulated in Table 5.16 were again varied. Perhaps most importantly, <u>no</u> subject proposed an interpretation of embolism/infarction for any of the three lung densities within the two-second viewing period, suggesting that this interpretation is not a highly salient interpretation among subjects for lung densities of the sort which appear in the film, at least not on the basis of what can be seen in a glance.

V.D.4 Pre-clinical Infarction/Embolism

Infarctions were present in the patient shown in Film 2. However, only six subjects gave an interpretation of embolism or infarction for any lung density in Film 2 prior to receiving clinical information, and of these only four maintained this interpretation as a final disposition. In this section, we will examine subjects' analyses made prior to receiving clinical data on the patient in an attempt to determine the basis for infarction and embolism interpretations. These analyses further illustrate the complexity of the mental processes of radiologists.

No subject had an interpretation of embolism/infarction during the two-second viewing period. Hence all of the interpretations to be discussed occurred either in the first full analysis of the film or in the subsequent first formal report. Most subjects initiated their first film analysis having activated at least a partial schema for the film.

Table 5.16
Major Initial Arguments – Film 2

	Failure	Cancer	Other Miscellaneous
1st / 2nd Year Residents			
RA1			X
RA2	X		
RA3			X
RA4			X
RA5		X	
RA6		X	
RA7		X	
RA8	X		
RA9			X
RA10		X	
RA11			X
3rd / 4th Year Residents			
RB1			X
RB2	X	X	
RB3		X	
RB4		X	
RB5		X	
RB6	X*		
RB7	X		
Experts			
E1		X	
E2		X	X
E3			
E4		X	
E5		X	

*Note, this subject did not state congestive heart failure directly but gave a pattern of interpretations (e.g., large heart, engorged hila, interstitial pulmonary edema) constent with and suggestive of heart failure.

Two residents (RA8, RB6) of the six subjects began their analyses of this film disposed toward heart failure arguments (see Table 5.16). Within the film analyses, subject RA8's primary interpretations of the lung densities were as edema related to heart failure. Additional interpretations of the densities as possibly embolism/infarction were based on the judgement that the densities were "fairly localized" (presumably in contrast to a more diffuse pattern the subject might expect to see for edema).

However, the subject abandoned embolism/infarction in favor or edema (other alternatives were metastasis, pneumonia) because, in the subject's judgement, infarctions "tend to be more peripheral (in the lungs)" than the more centrally located densities of this film. Similarly, subject RB6 interpreted two of the three densities as edema (or pneumonia). One density, however, was seen as different from the others in its features ("wedge-shaped") and its anatomical localization ("pleural-based"), and it was for these reasons that an interpretation of infarction was given and maintained for this one density. These features of lung densities ("conical" and pleural-based) are fairly characteristic of pulmonary infarctions radiologically (e.g. Lillington & Jamplis, 1977, p.151).

Three subjects (RB3, E1, E4) began their first full film analyses after having activated cancer schemas during the two-second viewing (see Table 5.16). These were based largely on the wide mediastinum in the film, with different degrees of involvement of other chest components. For all three of these subjects the chest evidence of thoracic surgery and/or what they interpreted to be mitral valvular problems (the valve replacement was actually aortic) was a major impetus for activating an alternative line of reasoning involving pulmonary infarction or embolism. For example, RB3, upon detecting the prosthetic valve (which he interpreted to be mitral), engaged the medical relationship between mitral valvular disease, subacute bacterial endocarditis, and septic emboli to establish embolism/infarction as an alternative interpretation to lung metastases for the lung densities of the film. However, for this resident, the cancer line of reasoning, based primarily on the degenerated arm bone and absent breast, overrode any alternatives and he concluded a general, wide-spread interpretation of cancer for the chest, including the lung densities as metastatic disease.

The two experts (E1, E4) also entered the first film analysis with an interpretation of mediastinally based cancer. For expert E1, the evidence of thoracic surgery activated a major alternative line of reasoning in which the mediastinum was interpreted not as cancer but as post-surgical mediastinal bleeding and the lung densities as post-surgical edema or infarction. Cancer was then confined only to the arm bone. In contrast to RB3, this expert proposed a two-process argument for the chest. Expert E4 maintained a wide-spread cancer argument throughout his analyses of the film. The interjection of embolism as a possible basis for (at least some) lung densities was based on two observations -- the mitral valvular association and a perceived discrepancy between the degree of density in the lung patches and the degree of density which would be expected under an interpretation of metastatic lesions.

The final subject (RA11) showed no discernible line of reasoning upon entering his first film analyses. "Ill-defined nodular densities in both lungs" were interpreted as "pneumonia or infarction, depending on the patient's clinical history." In support of these interpretations was the perception that the densities were "homogeneously opacified", perhaps in contrast to massed tumors.

Summary. Embolism/infarction was not a salient interpretation for the lung densities, as attested by the results that no subject gave this interpretation during two seconds and only six (of 23) subjects ever raised this interpretation prior to clinical information on the patient. Activation of this interpretation based on the film itself required opportunistic utilization of subtle film aspects including discriminations of shape (wedge) and density features, anatomical localization (pleural-based), abstracted medical arguments (surgical effects), and correspondences with heart valve abnormalities. Major lines of reasoning established in initial encoding were, again, largely detrimental in this film. However, in at least one instance, expert E4, the medical schema engaged by the subject provided criticism (through schema-driven expectations for features of lung densities) which led to generation of the correct alternative.

V.D.5 Resolution Of External Context

The clinical history provided to subjects with the film stated that the patient had experienced chest pain and shortness of breath, with no changes in temperature. These clinical history features are highly characteristic of infarction and embolism (e.g. Lillington & Jamplis, 1977, p.151). In fact, the number of subjects activating interpretations of embolism/infarction for the lung densities after the presentation of clinical data is in marked contrast to the number of subjects who raised this interpretation beforehand.*16 While only four residents (22%) advanced embolism/infarction before clinical data, ten residents (55%) did so afterward. The expert group included two subjects (40%) before clinical data and four subjects (80%) after clinical data were introduced. These interpretations were uniformly advanced in relation to the clinical information concerning chest pain and shortness of breath.

The general results from Film 2 provide a good view of the overall case resolution component of the radiological diagnosis process. In this film, the critical diagnosis information is not entirely within the film. Although diagnosticians vary in their ability to make discriminations within the film itself, resolution of their uncertainty is aided by a wide-ranging representation of the patient's anatomy and medical condition.

V.D.6 An Annotated Example

We illustrate the overall case resolution process by presenting a trace of the diagnosis offered by one subject, Expert E4. This trace shows in context many of the representational issues and mechanisms discussed in earlier sections presenting results from this film.

Two-second view. The subject's general impression of the film, based on the two-second viewing, was as lymphangitic (lymphatic system) spread of cancer - centered in the mediastinum, but quite widespread. In addition, other interpretations are made during the initial encoding and constraints are set which affect later analyses.

In the subject's initial statements, abnormality is localized in the mediastinal area, characterized, and interpreted as lymphangitic spread:

> All right. Mostly under-aerated lungs bilaterally (The subject has immediately posted a constraint that eliminates all of the hyper-expansive chest etiologies that were important in prior films discussed). A widened mediastinum (localization), but not in a clear-cut tumafactive impression (characterization that constrains out "mass" cancer arguments). It's more like a starburst (characterization that yields lymphangitic metastasis). This looks to me like lymphangitic cancer spread, certainly around the mediastinum. Primary I couldn't even guess at. The most common thing is probably carcinoma of the breast (the activated lymphangitic spread model provides clinical relationship to the breast condition, which the subject has not detected).

In discussing the lungs and hila, the two left-sided lung densities are merged within a single perception and tied into the lymphangitic spread. There is also an interesting suggestion that the characterization and interpretation of the these abnormalities is "composed" or "compiled" within a more unitary, automated operation:

> The hila were not well seen at all. They were covered by a rather irregular, multifingered density coming out from the hilus. It's the kind of thing you really can't describe as much as you can say. It looked to me like lymphangitic spread

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*16 Due to an error in consultation of the medical record for Film 2, residents were erroneously told that the patient was a male. The error was discovered before experts participated in the study. All other data were identical. The error might be expected to influence post-clinical considerations of cancer in various ways. The results reported in the present section concerning relationships of "chest pain" etc. to arguments of infarction/embolism are unlikely to have been affected by the error of gender. However, we feel that our error must be noted.

> coming out from the hila. Left lower lobe had a density in it that was fairly similar but not connected with (separated from) the hilus. I would say five centimeters in diameter.

The heart was evaluated and constraints set, tending to preclude heart failure and, more generally, cardiogenic arguments for the chest:

> The heart was also covered by the same thing (the "starburst" or "finger-like" condition). It's not the kind of pattern you see with congestive heart failure. It's not the kind of pattern you normally see with azotemia. Again, it looks like lymphangitic tumor.

<u>First analysis/report</u>. In the early parts of his first film analyses, the subject separated the two densities in the left lung, picked up the one on the right, and detected the prosthetic heart valve. There was a subsequent analysis of the heart which reaffirmed early impressions:

> ..The heart is moderately enlarged, not grossly so. This very definitely is not the picture of ordinary congestive heart failure...

Analysis then refocuses on the lung densities. Metastatic tumor congruent with the more general chest "picture" is the clearly predominant interpretation, but the door begins to open for interpretations of pulmonary emboli. The mechanisms which opportunistically intrude on the previously pat cancer argument involve feature re-characterization and medical arguments relating the prosthetic valve and the physiologic production of emboli:

> ..The multiple changes we see in the lungs would raise the question of multiple emboli and/or tumor. I would favor tumor because of the widened mediastinum that we're discussing. But I think we have two different things going on. It could be all tumor. It could not be all multiple emboli in any reasonable form. I think there is tumor here in both lungs. I wonder if we may not have a shower of emboli superimposed <u>simply because some of these are not as dense as tumor ought to be, and we do have an artificial cardiac valve, apparently a mitral valve</u>.

The disease model for lymphangitic metastasis now directs further looking for additional spread of the disease. Detected abnormalities in the arm and a rib reinforce the conception of cancer:

> In the periphery, there are some areas of not ordinary destruction: Irregular but rather well-defined demineralization in the right proximal humerus (the right

> arm). Not seen in other areas... I am now looking at the ribs which have to be looked at very carefully, and I don't see a portion of the right second rib - which is sneaky if I ever saw one, but this is the way they occur. You simply see the rib there and then there is a zone about three or four centimeters long in which it isn't there.

At this point the subject summarizes the film:

> OK. We're dealing with tumor destructive of the right rib involving the mediastinum. Lymphangitic spread into both lungs. Probably metastatic masses underneath the lymphangitic spread in both lungs. I would guess the changes in the right humerus are also probably due to metastases. The cardiac valve may be related only in that there is a possibility that there could be emboli superimposed on the rest of this. The basic pathology here is multiple tumors.

<u>After receiving clinical data</u>. Prior to a second analysis of the film, the subject was given the clinical history for the patient, which contained the characteristic clinical features of embolism. It can be seen that this information causes a definite shift of emphasis toward an embolism explanation. For many other subjects, clinical data was the initial impetus or "trigger" for <u>establishing</u> an embolism interpretation:

> All right. This is a 71 year old woman. Aortic valve replacement. Chest pain and shortness of breath. <u>Multiple emboli by clinical history</u>. No temperature change. <u>I still believe there is likely tumor underneath this</u>. Almost certainly tumor underneath this in the lungs as well as in the skeleton. But, I think the odds on pulmonary emboli has gone up abruptly.

Subsequent analyses of the film focus on reaffirming the presence, now of cancer, and on partitioning lesions between the multiple etiologies. This partitioning involves characterization of features yet again, but now this characterization is highly directed by the cancer and pulmonary embolism schemas:

> OK. Now knowing that the history is that of pulmonary emboli, some of these changes are very probably pulmonary emboli, particularly the one in the left mid-lung field. The one in the right is not as classical but it could be. I would still like to find out whether there is underlying tumor here and I badly want to find out what happened to this rib up here. I think there is a zone of ribs missing and we definitely have radiolucencies in the right lung which are probably tumor. So I still, I rather, I still believe that we have two diagnoses. We have pulmonary emboli and we have tumor. I think both of

them pretty definitive.

In summary of the lung densities:

There are also areas of increased density in both mid and lower lung fields, some of which are consistent with pulmonary emboli. At least two of these densities...(describes locations)..., are better defined than the usual pulmonary emboli and raise the distinct possibility of tumafaction.

As we have seen in prior discussions of Film 2, a primary problem for the diagnostician is apportioning the multiple abnormalities of the film according to etiology. The general tendency among subjects was to create uniform single-process arguments for the chest (although alternative but still <u>single</u>-process arguments were advanced by many subjects). Successful subjects utilized all the components of the diagnostic process (e.g., feature characterization, anatomical localization, medical explanation) to separate two processes that were manifest within the same chest.

CHAPTER VI

CONCLUDING OBSERVATIONS

Our broad conclusions from the work thus far completed are stated in Chapter I. They are summarized briefly in the points which follow.

- Detailed knowledge of anatomy is a critical aspect of expertise in X-ray film interpretation. Experts have the constructive perceptual ability to build a three-dimensional representation of the particular patient, adjusted to that patient's particular anatomical idiosyncrasies.

- The knowledge which underlies expertise includes the mental representation of anatomy, a theory of anatomic perturbation under pathology, and the projection of this anatomy into the radiographic domain. It appears that this internal representational knowledge takes years of experience to mature.

- The precise, rapid recognition skill that characterizes expertise is recursive. As experts notice new details in a film and as they confirm or rule out hypotheses, they continually refine the models they have constructed to represent the film's content. There appears to be a strong interaction between this recursive representational refinement and the broader general film encoding that experts also perform.

- Experts do more early constraint posting and deferring of final decisions. They have the flexibility to recursively pass through the diagnostic stages, and they are able to make highly accurate overall functional models of the chest systems to generate and organize the posted constraints.

- Expert radiologists are opportunistic planners with very rich recognition and constructive perceptual abilities. That is, they are very sensitive to relevant new information and know with some precision when to seek more data. Residents often order additional films and exotic tests in situations where experts can reach the correct diagnosis with assurance.

- o Novices are more likely to maintain bad film interpretations in the face of discrepant evidence from the patient's clinical history. Experts are more able to ignore data that are not relevant to a radiological diagnosis.

- o Expert-novice comparison work in several skill domains has characterized experts as high-speed classifiers or recognizers with detailed performance preprogrammed for each eventuality. It has been hypothesized that experts are primarily highly differentiated recognizers. Part of this argument is that expertise is primarily a highly differentiated recognition skill rather than a more complex general inferencing ability, and that recognition power comes from specific knowledge. On the other hand, as we look at very complex problem solving domains, such as radiological diagnosis, we see that the knowledge component of expertise is not limited to immediate recognitions. At the very least, expert recognitions recur throughout a period of representational construction that has much of the flavor of opportunistic planning.

There are two issues that deserve final comment. First, there is the question of the generality of our conclusions. Each film we analyzed produced a substantially different pattern of performance. While we saw common aspects to that performance, the issue of generality deserves some attention. The other major issue that merits discussion is the possible implications of the work we reported for instruction. Each of these issues is discussed below.

VI.A GENERALITY OF PROCESS ANALYSES

Radiological diagnosis arises from an interaction between the information content of the specific film and the knowledge base of the radiologist. The radiologist's knowledge base is a structure of schemas for constructing mental representations of anatomy, for recognizing abnormal film features, and for classifying and understanding the implications of disease conditions of patients. Because of the specificity of that knowledge, the specific ways in which the components of diagnosis are applied vary from one film to the next. This variation can be seen in the results we presented.

Single schema. Film 8 was a film for which most of the abnormal features (primarily those relating to the expansion of the lungs and the shape of the heart) were causally interconnected. The most salient abnormality (the sail-shaped density) should have triggered an explanatory schema that was consistent with the other abnormalities. Engagement of that schema could successfully drive the film analysis. If the schema were underdeveloped or faulty, the film interpretation would be less successful.

Schema cooperation. Film 9 was a film in which the primary film abnormality (the tumor) was causally and structurally disjoint from the other abnormal features of the film (which related to chronic obstructive pulmonary disease, or COPD). The COPD features were strongly interconnected causally and structurally, and the most salient film cues related to COPD. Hence, we would expect that engagement of the COPD schema might interfere with analysis of the tumor-related features. Success depended on both schemas being triggered and on the schemas being precise enough to accomplish their separate purposes without interfering with one another.

Schema ambiguity. Film 2 was one in which all of the abnormalities could have been subsumed under either of two or three schemas (cancer, heart failure, and surgical effects). In fact, though, different abnormalities manifested different specific problems. Engagement of any of the relevant schemas individually was detrimental to the successful analysis of the film. Success depended on opportunistic use of the few film cues that suggested the inadequacy of any single disorder, as well as information external to the film which could resolve the inherent ambiguity of the film itself.

Given this diversity of stimulus situations, in future work we will need to create and use exemplars from a taxonomy of films. This taxonomy will have to capture film features, causal and structural relationships, and the schema-triggering strength of features. These film characteristics can then be projected against the knowledge bases of individuals at different levels of experience. We must not, however, be premature in the specification of the varying course of the interaction between the film and the radiologist's mental processes. The specifics of individual performance are no doubt varied, and, especially in the expert, flexible and redundant. For instance, in Film 8, we do not know for sure whether (1) the triangle-shaped density triggered an atelectasis schema which then built a representation of the heart, lungs, etc.; (2) the lungs triggered hyperexpansion, which triggered atelectasis and heart expectations as effects; (3) the loss of heart border triggered a right middle lobe condition which generated atelectasis as one of a number of possibilities; or some other course of processing occurred.

All of these routes are plausible, and there is no compelling reason why we would expect a single means to a diagnostic end among experts. Constriction in means might, if anything, be presumed characteristic of novice processing. Experts may adapt to a task inherently high in ambiguity by flexibly mixing various components of the diagnostic process. Flexible use of processing resources does not imply chaos, either in the radiologist's approach or in our theorizing. Further studies of diagnostic problem solving involving stimulus classification and knowledge base documentation will constrain the range of plausible methods in a given type of film and subject.

Our analyses to date can be considered as descriptive of both general and more film-specific process components; both aspects are fundamental to a more complete account of diagnostic processing. The next step is to construct a model of processing activity and expert knowledge and then to carry out systematic tests of that model against a set of stimuli that is a representative sample of the relevant features of the expert's knowledge base and his or her processing capabilities.

VI.B IMPLICATIONS FOR INSTRUCTION

Two types of implications for instruction emerge from this work. First, the role of mental representation constructions has been shown to be substantial. As has been shown in other domains, expertise involves powerful problem representation abilities; in the present work, this has been manifested in the anatomical representation ability that is so critical to successful diagnosis. This finding has important implications for teaching. Second, the significance of constructed anatomical representations for diagnosis has implications for cost-benefit comparisons between increased use of diagnostic imaging aids and improved training arrangements.

Factual knowledge vs. understanding. In recent years, the medical school curriculum has moved away from traditional approaches to teaching anatomy to a greater concern with physiological anatomy, i.e., the functional interconnections of organs and the implications of structure for function. The mental representation abilities we found to be important are closer to surgical anatomy, which is concerned with finding one's way efficiently through the body--knowing what is behind the surfaces at which one is currently looking. This type of anatomical knowledge was thought to be provided by the dissections that are now less prevalent in medical school classes. The superficial interpretation of our results might be that more time should be given to such activities in the future.

However, a look at expertise in other domains may help us temper such conclusions and make them more useful. In the teaching of mathematics, there has been an oscillation between the teaching of computational skills (mathematics drills) and the teaching of the fundamental concepts that underly number and computation. "New math" movements that emphasize the meaning of numerical operations alternate with "back-to-basics" movements that emphasize efficiency of performance. What has been missing is the realization that close coupling of basic concepts with the specifics of performance is required for expertise. The same coupling of specific knowledge gained from experience with dissections, clinical practica (clerkships), and the like, with systematic fundamentals of body function and dysfunction is beginning to appear in medical education. There is much of importance in the functional anatomy that has replaced dissection-based training to some extent. What may be required is a combination of functional anatomy with more direct exposure to the spatial layout of anatomy and to its variations in different patients. We note that such "clinical correlations" are becoming more prevalent in medical education.

Another issue of medical education that also deserves brief comment is the increased likelihood that the resources will soon exist for substantial simulations of complex diagnostic cases. It should soon be quite feasible (to some extent it already is) for medical students to work at simulated diagnoses on the computer. If such diagnostic exercises are to be maximally useful, it will be necessary for the computer to have its own model of the state of the student's diagnostic knowledge. This will permit intelligent hinting and selection of problems to maximize their instructional effect. Such modeling capability will be improved by extensions of the present work into a more formal model of the diagnostic process, an activity we have now begun.

<u>Trading off instructional and equipment costs</u>. A second instructional issue to be raised concerns the possibility of using models of diagnostic processing to inform decision making about the use of sophisticated imaging equipment to assist in the diagnostic process. We note specifically that what was missing in our resident radiologists, who had already had substantial training, was the ability to construct a representation of the patient's anatomy from the minimal information in a chest X-ray picture. As our knowledge of the limits on this capability increases and as we better specify which aspects of the skill are acquired only with long experience, it may be possible to propose clear economic tradeoff rules that tell us how reliability can be increased and how training costs can be decreased by the introduction of specific diagnostic imaging aids. This could be an improvement over the current situation in which the monetary costs of devices such as computerized axial tomography and nuclear magnetic resonance equipment are clear while the benefits in improved reliability or decreased training requirements are harder to quantify.

APPENDIX A

REFERENCES

Blesser, B., & Ozonoff, D. A model for the radiologic process. Radiology, 1972, 103, 515-521.

Carmody, D. P., Nodine, C. F., & Kundel, H. L. Global and segmented search for lung nodules of different edge gradients. Investigative Radiology, 1980, 15(3), 224-233.

Christensen, E. E., Dietz, G. W., Murry, R. C., & Moore, J. G. The effect of fatigue on resident performance. Radiology, 1977, 125, 103-105.

Christensen, E. E., Murry, R. C., Holland, K., Reynolds, J., Landay, M.J., Moore, J. G. The effect of search time on perception. Radiology, 1981, 138, 361-365.

Feltovich, P. J. Knowledge-based components of expertise in medical diagnosis. Unpublished doctoral dissertation, University of Minnesota, 1981.

Garland, L. H. Studies on the accuracy of diagnostic procedures. American Journal of Roentgenology, 1959, 82, 25-38.

Goodenough, D. J., Rossmann, K., & Lusted, L. B. Factors affecting the detectability of a simulated radiographic signal. Investigative Radiology, 1973, 8(5), 339-344.

Gray, J. E., Taylor, K. W., & Hobbs, B. B. Detection accuracy in chest radiography. American Journal of Roentgenology, 1978, 131, 247-253.

Hallberg, J. R., Kelsey, C. A., & Briscoe, D. Some effects of method on the measured conspicuity of chest lesions. Investigative Radiology, 1978, 13(5), 439-443.

Hayes-Roth, B., & Hayes-Roth, F. A cognitive model of planning. Cognitive Science, 1979, 3, 275-310.

Heitzman, E. R. The lungs: Radiologic-pathologic correlations. St. Louis, MO: Mosby, 1973.

Herman, P. G., Gerson, D. E., Hessel, S. J., Mayer, B. S., Watnick, M., Blesser, B., & Ozonoff, D. Disagreements in chest roentgen interpretation. Chest, 1975, 68(3), 278-282.

Johnson, P. E., Barreto, A., Hassebrook, F., Moller, J., Prietula, M., Feltovich, P. J., & Swanson, D. B. Expertise and error in diagnostic reasoning. Cognitive Science, in press.

Kundel, H. L. Visual sampling and estimates of the location of information on chest films. Investigative Radiology, 1974, 9(2), 87-93.

Kundel, H. L. Predictive value and threshold detectability of lung tumors. Radiology, 1981, 139, 25-29.

Kundel, H. L., & La Follette, P. S. Visual search patterns and experience with radiological images. Radiology, 1972, 103, 523-528.

Kundel, H. L., & Nodine, C. F. Interpreting chest radiographs without visual search, Radiology, 1975, 116, 527-532.

Kundel, H.L., Nodine, C.F., & Carmody D. Visual scanning, pattern recognition and decision-making in pulmonary nodule detection. Investigative Radiology, 1978, 13, 175-181.

Kundel, H. L., & Revesz, G. Conspicuity effects in chest radiography. Proceedings of the Symposium on Optimization of Chest Radiography, University of Wisconsin, 1979. HHS Publication (FDA), 80-8124, 1980.

Kundel, H. L., Revesz, G., & Toto, L. Contrast gradient and the detection of lung nodules. Investigative Radiology, 1979, 14(1), 18-22.

Kundel, H. L., & Wright, D. J. The influence of prior knowledge on visual search strategies during the viewing of chest radiographs. Radiology, 1969, 93, 315-320.

Lillington, G. A., & Jamplis, R. W. A diagnostic approach to chest diseases: Differential diagnosis based on roentgenographic patterns. Baltimore, MD: The Williams and Wilkins Co., 1977.

Meehan, J. R. (Ed.) The new UCI LISP manual. Hillsdale, NJ: Lawrence Erlbaum Associates, Inc., 1979.

Meschan, I. Synopsis of analysis of Roentgen signs in general radiology, Phil., PA: W. B. Saunders Co., 1976.

Rabin, C. B., & Baron, M. G. Radiology of the chest, 2nd edition. Baltimore, MD: Williams and Wilkens, Co., 1980.

Ravin, C. E. Disease signs in chest radiography. Proceedings of Symposium on Optimization of Chest Radiography, University of Wisconsin, 1979. HHS Publication (FDA) 80-8124, 1980.

Revesz, G., & Kundel, H. L. Psychophysical studies of detection errors in chest radiology. Radiology, 1977, 123, 559-562.

Revesz, G., Kundel, H. L., & Graber, M. A. The influence of structured noise on the detection of radiologic abnormalities. Investigative Radiology, 1974, 9(6), 479-486.

Sandor T., & Swensson, R. G. Evaluation of observer performance in detecting blood vessels on simulated angiographic images. Medical Physics, 1978, 5(5), 380-386.

Schreiber, M. H. The clinical history as a factor in roentgenogram interpretation. Journal of the American Medical Association, 1963, 185(5), 399-401.

Smith, G. R., van Belle, G., & Loop, J. W. Pattern discrimination in matching chest radiographs. Radiology, 1978, 127, 595-599.

Smith, M. J. Error and variation in diagnostic radiology. Springfield, MA: Charles C. Thomas, 1967.

Starr, S. J., Metz, C. E., Lusted, L. B., & Goodenough, D. J. Visual detection and localization of radiographic images. Radiology, 1975, 116, 533-538.

Stefik, M. Planning with constraints (MOLGEN: Part 1), Artificial Intelligence, 1981, 16, 111-169. (a)

Stefik, M. Planning and Meta-Planning (MOLGEN: Part 2). Artificial Intelligence, 1981, 16, 111-169. (b)

Sussman, G. J. A computer model of skill acquisition. New York: American Elsevier Publishing Company, Inc., 1975.

Swensson, R. G. A two-stage detection model applied to skilled visual search by radiologists. Perception & Psychophysics, 1980, 27(1), 11-16.

Swensson, R. G., Hessel, S. J., & Herman, P. G. Omissions in radiology: Faulty search or stringent reporting criteria? Radiology, 1977, 123, 563-567.

Swensson, R. G., Hessel, S. J., & Herman, P. G. Detection before attention in skilled visual search. Paper presented at the annual meeting of the Psychonomic Society, November 1978.

Swensson, R. G., Hessel, S. J., & Herman, P. G. Detection performance and the nature of the radiologist's search task. Proceedings of the Symposium on the Optimization of Chest Radiology, University of Wisconsin, 1979. HHS Publication (FDA) 80-8124, 1980.

Taber, F. Visual perception of roentgen images. CRC Critical Reviews in Diagnostic Imaging, 1977, 187-202.

Thomas, E. L. Search behavior. Radiologic Clinics of North America, 1969, 7(3), 403-417.

Thomas, E. L., & Lansdown, E. L. Visual search patterns of radiologists in training. Radiology, 1963, 81, 288-292.

Tuddenham, W. J. Visual search, image organization, and reader error in roentgen diagnosis. Radiology, 1962, 78, 694-704.

Tuddenham, W. J. Problems of perception in chest roentgenology: Facts and fallacies. In B. Felson (Ed.), Proceedings of The Radiologic Clinics of North America Symposium on Diagnostic Roentgenology of the Chest. 1963, 1(2), 277-289.

Tuddenham, W. J. Visual physiology and instrumentation in roentgen diagnosis. Diagnostic Radiologic Instrumentation, Springfield, IL: C.C. Thomas, 1965.

Tuddenham, W. J., & Calvert, W. P. Visual search patterns in roentgen diagnosis. Radiology, 1961, 76, 255-256.

Yerushalmy, J. The statistical assessment of the variability in observer perception and description of roentgenographic pulmonary shadows. Radiologic Clinics of North America, 1969, 7(3), 381-392.

APPENDIX B

GLOSSARY

aneurysm - a localized, abnormal dilatation (expansion) of a blood vessel due to a congenital defect or weakness of the wall of the vessel

aorta - the major heart vessel leading from the left ventricle of the heart to all body parts except the lungs.

aortic valve - the heart valve between the left ventricle of the heart and the aorta.

apex - the uppermost region of the lung; the lower-left region (pointed area) of the heart, composed of ventricular musculature.

atelectasis - a collapsed or airless condition of the lung (see text)

atherosclerosis - a form of arteriosclerosis (ie. hardening and loss of elasticity of the blood vessels, especially the arteries) which involves localized accumulation of lipid (fatty) materials within or beneath the blood vessel, resulting in poor blood circulation.

azotemia - presence of excess nitrogenous bodies in the blood as a result of kidney insufficiency

bleb - a small air cyst (sac)

bronchi - the two main branches leading from the trachea* which provide the passageway for air moving to and from the lungs.

bronchitis - inflammation of the lining of the bronchi*

bronchogenic carcinoma - malignant growth originating in the bronchi*, which tends to infiltrate and give rise to metastases*

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* These words are also defined in this glossary.

bulla - large blister or cyst filled with air or fluid (bullae - plural; bullous - adjective)

carcinoma - cancer; a new growth which tends to infiltrate and give rise to metastasis*

cardiac - pertaining to the heart

Chronic Obstructive Pulmonary Disease - generalized airways obstruction causing decreased ability of the lungs to perform their function of ventilation. Condition appears in a number of diseases including emphysema, bronchitis and chronic asthma.

congestive heart failure - a condition in which there is reduced ability of the heart to supply blood. Usually associated with blood stagnation.

COPD - see Chronic Obstructive Pulmonary Disease

cor pulmonale - heart disease secondary to disease of the lungs, involving failure or thickening (hypertrophy) of the right ventricle (a chamber of the heart).

demineralization - loss of calcium and phosphorus salts in the bones by excessive absorption or excretion

diaphragm - the muscular structure which forms a partition between the chest and the abdomen

edema - swelling; a condition in which the body tissues contain an excessive amount of body fluid. Edema may be localized or general (sometimes called "dropsy")

emboli -blockage or occlusion of a blood vessel by a fragment of a blood clot, an air bubble, or other foreign body

embolus - singular of emboli

emphysema - a condition in which areas of the lungs become distended (overly expanded) or ruptured. Usually the result of airways obstruction, which hinders expiration (exhaling), and/or loss of elasticity in the lung.

endocarditis - inflammation of the membrane lining the inner surface and cavities of the heart

esophagus - a muscular tube for the passage of food which runs from the neck region to the stomach (a/k/a "gullet") fibrotic - a condition marked by the formation of fibers, often resulting from a previous acute (eg. inflammatory) process.

fibrous - composed of or containing fibers.

fissure - a groove or natural division between organs, eg. lung fissures connote the separation between individual lobes of the lung, as in the minor fissure*

G-I - *abbrev for gastro-intestinal;* pertaining to the stomach and small intestine (a/k/a "the gut")

hemidiaphragm - the right or left side of the diaphragm*

hemithorax - one side of the chest; eg. the right hemithorax

hilar - pertaining to the hilum*

hilum - the region where the bronchi* and great vessels of the heart feed into and out of the lung; the roots of the lungs where they join the mediastinum*

humeral - pertaining to the humerus*

humerus - upper bone of the arm from the elbow to the shoulder joint

hypoplastic - defective or incomplete development; eg. hypoplastic lung - smaller than usual, underdeveloped lung

infarct - region of necrosis* (dead tissue) and hemorrhage in an organ resulting from obstruction of the local circulation by, for example, an embolus*

infiltrate - invasion a tissue or organ by a substance not normal to it; eg. infectious or neoplastic processes, fluid.

infra- prefix meaning "below, under or beneath" [ie. inferior to] which is used to localize a feature relative to an anatomical landmark. eg. infrahilar = located below the hilum

lymphangitic metastasis - metastasis* of cancer via the lymph system

lymphoma - general term for growth of new tissue in the lymphatic system; eg. Hodgkin's disease

mediastinum - the middle compartment of the chest containing the trachea*, esophagus*, the heart and the great vessels

metastasis - invasion of cancer cells from one location of the body to another. Metastatic spread is by the blood stream or lymph system.

minor fissure -one of the fissures* of the lung found in the right chest, separating the right middle and right upper lobes. Normally runs in a perpendicular direction relative to the spine.

mitral stenosis - constriction or narrowing of the mitral valve * and/or orifice

mitral valve - the cardiac valve which separates the left atrium and the left ventricle of the heart.

necrosis - death of areas of tissue or bone surrounded by healthy parts. eg. necrosis due to cessation of blood supply = infarct*

node - a small rounded organ or structure; eg. lymph node

nodule - a small node* or a small aggregation of cells

osteoporosis - increased porosity of bone tissue with softening of bone tissue; the demineralization* of the bone makes it more permeable to X-rays, thus the appearance of these areas are darker than normal calcified bone

para- prefix meaning "near or beside" used in conjunction with an anatomical landmark; eg. paravertebral = near the vertebrae (spinal cord)

parenchyma - the outer functioning portion of the lungs containing the air sacs

peri- prefix meaning "around or about" some area; eg. perihilar haziness = haziness around the hilar region

pleura - membrane covering the lung and lining of the chest

pleural - pertaining to the pleura*

pleural effusion - fluid filling the membrane covering the lung and lining of the chest (see pleura*)

prosthesis - an artificial device to replace a missing or diseased part of the body; eg. prosthetic mitral valve*

prosthetic aortic valve - an artificial (prosthesis*) valve placed in lieu of a malfunctioning aortic valve (see valvular disease*)

pulmonary - relating to the lungs

pulmonary venous hypertension - increased pressure in the pulmonary veins; often associated with heart failure or obstruction to flow in the left side of the heart.

reticular - having the appearance of a network; eg. reticular nodular

sarcoidosis - a chronic infectious disease of unknown origin which causes circumscribed lesions in the lungs (as well as in other tissues)

septic - pertaining to sepsis, the presence of pathogenic organisms, (eg. bacteria) or their toxins in the blood or other tissues.

supra- prefix meaning "above" [ie. superior to] which is used to localize a feature relative to an anatomical landmark. eg. suprahilar = above the hilum

thoracic - pertaining to the thorax* or the chest

thorax - the chest

tortuous - a winding, twisting, abnormal contour; eg. tortuous aorta* = a twisting aorta* seen most often in older individuals

trachea - a thin-walled tube made of cartilege which runs from the head region to the bronchi* carrying air to the lungs (a/k/a "the windpipe")

uncoiled aorta - see tortuous*

valvular disease - disease of the valves of the heart which disrupt the normal flow of blood into and out of the heart; eg. mitral stenosis*

vascularity - blood vessels

APPENDIX C

EXAMPLES OF PROTOCOL SCORINGS (FILM 8)

C.A FIRST YEAR RESIDENTS

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First Year Subject RA1

*** 2 SEC ***
------ EFFECT 0 — 0 INCR_DENS/BULGE/PROM/SUSPICIOUS_AREA_R_HT_BORD_
                      OR_PERICARD/PARASPINAL
CAUSE  ------ 7 — 9 ECTATIC_OR_TORTUOUS_OR_UNFOLDED_AORTA

INCR_DENS/BULGE/PROM/SUSPICIOUS_AREA_R_HT_BORD_OR_PERICARD/PARASPINAL
     EXPLAINED BY
          ECTATIC_OR_TORTUOUS_OR_UNFOLDED_AORTA

*** PROMPT ***
------ ------ 3 — 0 R_PERICARDIAL/HT_BORDER_INCR_DENS

*** REPORT 1 ***
------ EFFECT 3 — 3
TRIANGULAR_DENS_R_MID_LUNG_MEDIALLY_HT_BORD/PARASPINAL
------ EFFECT 3 — 0 POORLY_SEEN_R_HILUM
CAUSE  ------ 3 — 0 INFERIORLY_PULLED_R_HILUM
------ ------ 5 — 6 DECR_VASC_R_LUNG
------ ------ 5 — 0 DECR_VASC_R_LUNG_APEX
CAUSE  ------ 1 — 0 MEDIAL_R_MID_LUNG_MASS
CAUSE  ------ 1 — 0 ATELECTASIS_R_MID_LUNG_MEDIALLY
CAUSE  ------ 5 — 5 INCR_EXPANSION_R_LUNG
------ EFFECT 5 — 5 INCR_LUCENCY_R_LUNG

INCR_LUCENCY_R_LUNG
     EXPLAINED BY
          INCR_EXPANSION_R_LUNG

POORLY_SEEN_R_HILUM
     EXPLAINED BY
          INFERIORLY_PULLED_R_HILUM

TRIANGULAR_DENS_R_MID_LUNG_MEDIALLY_HT_BORD/PARASPINAL
     EXPLAINED BY
          MEDIAL_R_MID_LUNG_MASS
          OR ATELECTASIS_R_MID_LUNG_MEDIALLY

*** REPORT 2 ***
------ ------ 3 — 3 TRIANGULAR_DENS_R_MID_LUNG_MEDIALLY_HT_
                      BORD/PARASPINAL
------ EFFECT 5 — 5 INCR_LUCENCY_R_LUNG
CAUSE  ------ 5 — 6 DECR_VASC_R_LUNG
CAUSE  ------ 5 — 5 R_LUNG_EMPHYSEMA

INCR_LUCENCY_R_LUNG
     EXPLAINED BY
          DECR_VASC_R_LUNG
          AND R_LUNG_EMPHYSEMA
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First Year Subject RA2

*** 2 SEC ***
------ EFFECT 2 — 3 TRIANGULAR/WEDGED/SHARP_DENS_R_HT_BORD/PERICARDIAL
CAUSE  ------ 2 — 0 ATELECTASIS_R_HT_BORD_AREA
------ ------ 9 — 0 OLD_AGE
------ EFFECT 5 — 0 STRINGY_DENS_BOTH_LUNGS
CAUSE  ------ 5 — 5 CHRONIC_INTERSTITIAL_OR_FIBROTIC_DISEASE_BOTH_
                      LUNGS
------ ------ 9 — 0 INCR_STOMACH_AIR
------ ------ 5 — 4 POORLY_SEEN_RT_HEART_BORDER

STRINGY_DENS_BOTH_LUNGS
     EXPLAINED BY
          CHRONIC_INTERSTITIAL_OR_FIBROTIC_DISEASE_BOTH_LUNGS

TRIANGULAR/WEDGED/SHARP_DENS_R_HT_BORD/PERICARDIAL
     EXPLAINED BY
          ATELECTASIS_R_HT_BORD_AREA

*** PROMPT ***
CAUSE  EFFECT 2 — 3 TRIANGULAR/WEDGED/SHARP_DENS_R_HT_BORD/PERICARDIAL
------ EFFECT 5 — 4 POORLY_SEEN_RT_HEART_BORDER
CAUSE  ------ 2 — 0 ATELECTASIS_R_HT_BORD_AREA

POORLY_SEEN_RT_HEART_BORDER
     EXPLAINED BY
          TRIANGULAR/WEDGED/SHARP_DENS_R_HT_BORD/PERICARDIAL

TRIANGULAR/WEDGED/SHARP_DENS_R_HT_BORD/PERICARDIAL
     EXPLAINED BY
          ATELECTASIS_R_HT_BORD_AREA

*** REPORT 1 ***
------ ------ 5 — 5 INCR_LUCENCY_R_LUNG
CAUSE  EFFECT 5 — 5 BULLAE_R_UPPER_LOBE
CAUSE  EFFECT 5 — 5 BULLAE_R_LOWER_LOBE
------ EFFECT 2 — 3 TRIANGULAR/WEDGED/SHARP_DENS_R_HT_BORD/PERICARDIAL
CAUSE  EFFECT 1 — 1 R_MID_LOBE_ATELECTASIS
CAUSE  ------ 5 — 5 EMPHYSEMA_R_UPPER_LOBE
CAUSE  ------ 5 — 0 EMPHYSEMA_R_LOWER_LOBE
------ EFFECT 5 — 7 POORLY_SEEN_R_MEDIAL_DIAPHRAGM
CAUSE  EFFECT 3 — 2 R_LOWER_LOBE_ATELECTASIS

BULLAE_R_UPPER_LOBE
AND BULLAE_R_LOWER_LOBE
     EXPLAINED BY
          EMPHYSEMA_R_UPPER_LOBE
          AND EMPHYSEMA_R_LOWER_LOBE

R_MID_LOBE_ATELECTASIS
     EXPLAINED BY
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          BULLAE_R_UPPER_LOBE
          AND BULLAE_R_LOWER_LOBE

R_LOWER_LOBE_ATELECTASIS
     EXPLAINED BY
          BULLAE_R_UPPER_LOBE
          AND BULLAE_R_LOWER_LOBE

TRIANGULAR/WEDGED/SHARP_DENS_R_HT_BORD/PERICARDIAL
     EXPLAINED BY
          R_MID_LOBE_ATELECTASIS

POORLY_SEEN_R_MEDIAL_DIAPHRAGM
     EXPLAINED BY
          R_LOWER_LOBE_ATELECTASIS

*** REPORT 2 ***
------ ------ 5 -- 5 INCR_LUCENCY_R_LUNG
CAUSE  EFFECT 5 -- 5 BULLAE_R_UPPER_LOBE
CAUSE  EFFECT 5 -- 5 BULLAE_R_LOWER_LOBE
------ EFFECT 1 -- 1 R_MID_LOBE_ATELECTASIS
------ EFFECT 3 -- 2 R_LOWER_LOBE_ATELECTASIS
CAUSE  ------ 5 -- 5 R_LUNG_EMPHYSEMA
CAUSE  EFFECT 5 -- 5 CHRONIC_INTERSTITIAL_OR_FIBROTIC_DISEASE_R_LUNG
BULLAE_R_UPPER_LOBE
AND BULLAE_R_LOWER_LOBE
     EXPLAINED BY
          CHRONIC_INTERSTITIAL_OR_FIBROTIC_DISEASE_R_LUNG

R_MID_LOBE_ATELECTASIS
AND R_LOWER_LOBE_ATELECTASIS
     EXPLAINED BY
          BULLAE_R_UPPER_LOBE
          AND BULLAE_R_LOWER_LOBE

CHRONIC_INTERSTITIAL_OR_FIBROTIC_DISEASE_R_LUNG
     EXPLAINED BY
          R_LUNG_EMPHYSEMA
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First Year Subject RA3

*** 2 SEC ***
------ ------ 7 -- 0 INCR_DENS_BOTH_LUNGS
------ ------ 3 -- 3 R_HT_BORD_OR_PERICARD_MASS

*** PROMPT ***
------ EFFECT 7 -- 0 INCR_DENS_BOTH_LUNGS
CAUSE  ------ 5 -- 0 INCR_INTERSTIT_MARK_BOTH_LUNGS
CAUSE  ------ 3 -- 3 R_PARASPINAL_MASS
CAUSE  ------ 3 -- 3 R_HT_BORD_OR_PERICARD_MASS
------ EFFECT 0 -- 0 INCR_DENS/BULGE/PROM/SUSPICIOUS_AREA_R_HT_BORD_
                       OR_PERICARD/PARASPINAL

INCR_DENS_BOTH_LUNGS
     EXPLAINED BY
          INCR_INTERSTIT_MARK_BOTH_LUNGS

INCR_DENS/BULGE/PROM/SUSPICIOUS_AREA_R_HT_BORD_OR_PERICARD/PARASPINAL
     EXPLAINED BY
          R_PARASPINAL_MASS
          OR R_HT_BORD_OR_PERICARD_MASS

*** REPORT 1 ***
------ ------ 7 -- 0 INCR_VASC_BOTH_LUNGS
------ ------ 7 -- 9 INCR_VASC_L_LUNG
------ ------ 9 -- 9 RETICULAR_NODULAR_PATT_L_LUNG
------ EFFECT 9 -- 0 ATYPICAL_LUNG_PATT_R_LUNG
CAUSE  ------ 8 -- 9 UNUSUAL_OR_SPLAYED_OR_PROM_OR_CROWDED_VASC_R_LUNG
------ ------ 7 -- 0 POORLY_SEEN_RT_DIAPHRAGM
------ ------ 5 -- 0 R_PERICARDIAL_FAT_PAD
------ ------ 5 -- 4 POORLY_SEEN_RT_HEART_BORDER
------ EFFECT 3 -- 0 SHARP_MASS_LOWER_MID_R_PARASPINAL
CAUSE  ------ 8 -- 0 PULMONARY_OUTFLOW_TRACT_LOWER_MID_R_PARASPINAL
CAUSE  ------ 2 -- 0 TUMOR_LOWER_MID_R_PARASPINAL
CAUSE  ------ 9 -- 9 ABNORMAL_HEART_CONFIGURATION
CAUSE  ------ 3 -- 0 CANCEROUS_MASS_LOWER_MID_R_PARASPINAL
CAUSE  ------ 8 -- 0 ATYPICAL_VASC_LOWER_MID_R_PARASPINAL

ATYPICAL_LUNG_PATT_R_LUNG
     EXPLAINED BY
          UNUSUAL_OR_SPLAYED_OR_PROM_OR_CROWDED_VASC_R_LUNG

SHARP_MASS_LOWER_MID_R_PARASPINAL
     EXPLAINED BY
          PULMONARY_OUTFLOW_TRACT_LOWER_MID_R_PARASPINAL
          OR TUMOR_LOWER_MID_R_PARASPINAL
          OR ABNORMAL_HEART_CONFIGURATION
          OR CANCEROUS_MASS_LOWER_MID_R_PARASPINAL
          OR ATYPICAL_VASC_LOWER_MID_R_PARASPINAL

*** REPORT 2 ***
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------ EFFECT 0 — 7 ATYPICAL_LUNG_PATT_BOTH_LUNGS
CAUSE  EFFECT 0 — 9 INCR_DENS_L_LUNG
CAUSE  ------ 7 — 9 INCR_VASC_L_LUNG
CAUSE  ------ 9 — 9 RETICULAR_NODULAR_PATT_L_LUNG
CAUSE  ------ 8 — 9 UNUSUAL_OR_SPLAYED_OR_PROM_OR_CROWDED_VASC_R_LUNG
CAUSE  ------ 9 — 9 ABNORMAL_HEART_CONFIGURATION
------ EFFECT 3 — 3 SHARP_R_PARASPINAL_MASS
CAUSE  ------ 0 — 3 R_PARASPINAL_TUMOR
CAUSE  ------ 0 — 6 R_PARASPINAL_ATYPICAL_VASC

INCR_DENS_L_LUNG
     EXPLAINED BY
          INCR_VASC_L_LUNG
          OR RETICULAR_NODULAR_PATT_L_LUNG

ATYPICAL_LUNG_PATT_BOTH_LUNGS
     EXPLAINED BY
          INCR_DENS_L_LUNG
          AND UNUSUAL_OR_SPLAYED_OR_PROM_OR_CROWDED_VASC_R_LUNG

SHARP_R_PARASPINAL_MASS
     EXPLAINED BY
          R_PARASPINAL_TUMOR
          OR R_PARASPINAL_ATYPICAL_VASC
          OR ABNORMAL_HEART_CONFIGURATION
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 First Year Subject RA4

*** 2 SEC ***
------ ------ 9 -- 0 OLD_AGE
------ ------ 5 -- 5 CHRONIC_INTERSTITIAL_OR_FIBROTIC_DISEASE_BOTH_
                       LUNGS
------ ------ 3 -- 0 R_PERICARDIAL/HT_BORDER_INCR_DENS

*** PROMPT ***
------ ------ 5 -- 5 CHRONIC_INTERSTITIAL_OR_FIBROTIC_DISEASE_BOTH_
                       LUNGS
------ EFFECT 3 -- 0 R_PERICARDIAL/HT_BORDER_INCR_DENS
CAUSE  ------ 3 -- 0 R_LUNG_ATELECTASIS
CAUSE  ------ 0 -- 0 R_VENTRICLE
CAUSE  ------ 0 -- 0 R_ATRIUM

R_PERICARDIAL/HT_BORDER_INCR_DENS
     EXPLAINED BY
          R_LUNG_ATELECTASIS
          OR R_VENTRICLE
          OR R_ATRIUM

*** REPORT 1 ***
------ EFFECT 3 -- 0 R_MID_LUNG_HT_BORD_MASS
CAUSE  ------ 2 -- 0 TUMOROUS_MASS_R_MID_LUNG
CAUSE  ------ 1 -- 1 R_MID_LOBE_ATELECTASIS
------ ------ 9 -- 9 SURGICAL_STAPLES_R_LATERAL_LUNG
------ ------ 9 -- 9 PROM_L_HILUM
------ ------ 9 -- 9 L_HILAR_LESION
------ ------ 5 -- 5 CHRONIC_INTERSTITIAL_OR_FIBROTIC_DISEASE_BOTH_
                       LUNGS

R_MID_LUNG_HT_BORD_MASS
     EXPLAINED BY
          TUMOROUS_MASS_R_MID_LUNG
          OR R_MID_LOBE_ATELECTASIS

*** REPORT 2 ***
------ ------ 9 -- 9 SURGICAL_STAPLES_R_LATERAL_LUNG
------ ------ 3 -- 3 R_HT_BORD_OR_PERICARD_MASS
------ ------ 9 -- 9 PROM_L_HILUM
------ EFFECT 9 -- 9 L_HILAR_LESION
CAUSE  ------ 0 -- 0 LYMPH_NODE
CAUSE  ------ 0 -- 0 PULMONARY_ARTERY
------ ------ 5 -- 5 CHRONIC_INTERSTITIAL_OR_FIBROTIC_DISEASE_BOTH_
                       LUNGS

L_HILAR_LESION
     EXPLAINED BY
          LYMPH_NODE
          OR PULMONARY_ARTERY
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First Year Subject RA5

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*** 2 SEC ***
------ ------ 9 — 9 ABNORMAL_HEART_CONFIGURATION
------ ------ 7 — 0 FIBROSIS_BOTH_LUNGS

*** PROMPT ***
------ EFFECT 5 — 5 CHRONIC_INTERSTITIAL_OR_FIBROTIC_DISEASE_BOTH_
                      LUNGS
CAUSE  ------ 9 — 0 OLD_AGE
------ ------ 9 — 9 ABNORMAL_HEART_CONFIGURATION

CHRONIC_INTERSTITIAL_OR_FIBROTIC_DISEASE_BOTH_LUNGS
     EXPLAINED BY
          OLD_AGE

*** REPORT 1 ***
------ ------ 7 — 0 CHRONIC_INTERSTITIAL_OR_FIBROTIC_DISEASE_L_LUNG
------ ------ 5 — 5 INCR_LUCENCY_R_LUNG
------ ------ 7 — 5 R_INTERSTITIAL_HONEYCOMB_PATT

*** REPORT 2 ***
------ ------ 5 — 5 CHRONIC_INTERSTITIAL_OR_FIBROTIC_DISEASE_BOTH_
                      LUNGS
------ ------ 5 — 5 INCR_LUCENCY_R_LUNG
------ ------ 7 — 5 R_INTERSTITIAL_HONEYCOMB_PATT
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First Year Subject RA6

*** 2 SEC ***
------ EFFECT 2 -- 3 TRIANGULAR/WEDGED/SHARP_DENS_R_HT_BORD/PERICARDIAL
CAUSE  ------ 3 -- 0 R_HILAR_ABNORMALITY
------ EFFECT 9 -- 0 POORLY_SEEN_L_LUNG
CAUSE  ------ 9 -- 0 IMPROPER_PATIENT_POSITION/_PART_EXCLUDED_FROM_FILM
------ ------ 9 -- 0 POORLY_SEEN_L_COSTOPHRENIC_ANGLE

TRIANGULAR/WEDGED/SHARP_DENS_R_HT_BORD/PERICARDIAL
     EXPLAINED BY
          R_HILAR_ABNORMALITY

POORLY_SEEN_L_LUNG
     EXPLAINED BY
          IMPROPER_PATIENT_POSITION/_PART_EXCLUDED_FROM_FILM

*** PROMPT ***
------ EFFECT 9 -- 0 POORLY_SEEN_L_LUNG
CAUSE  ------ 9 -- 0 IMPROPER_PATIENT_POSITION/_PART_EXCLUDED_FROM_FILM
------ ------ 3 -- 0 R_HILAR_TRIANGULAR_DENS
------ ------ 7 -- 0 POORLY_SEEN_L_DIAPHRAGM
------ ------ 9 -- 0 POORLY_SEEN_L_COSTOPHRENIC_ANGLE

POORLY_SEEN_L_LUNG
     EXPLAINED BY
          IMPROPER_PATIENT_POSITION/_PART_EXCLUDED_FROM_FILM

*** REPORT 1 ***
------ EFFECT 2 -- 0 R_MID_LUNG_HT_BORD_OR_PARASPINAL_TRIANGULAR_DENS
CAUSE  ------ 0 -- 0 R_HILAR_ORIGIN
CAUSE  ------ 1 -- 0 R_MID_LOBE_LESION

R_MID_LUNG_HT_BORD_OR_PARASPINAL_TRIANGULAR_DENS
     EXPLAINED BY
          R_HILAR_ORIGIN
          AND R_MID_LOBE_LESION

*** REPORT 2 ***
------ EFFECT 3 -- 3 R_LUNG_TRIANGULAR_DENS
CAUSE  ------ 0 -- 0 R_HILAR_ORIGIN
CAUSE  ------ 0 -- 0 R_MID_LOBE

R_LUNG_TRIANGULAR_DENS
     EXPLAINED BY
          R_HILAR_ORIGIN
          AND R_MID_LOBE
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First Year Subject RA7

*** 2 SEC ***
------ ------ 3 -- 0 ABNORMAL_R_INFRA_HILUM
------ ------ 5 -- 5 CHRONIC_INTERSTITIAL_OR_FIBROTIC_DISEASE_BOTH_
                      LUNGS

*** PROMPT ***
------ ------ 5 -- 5 CHRONIC_INTERSTITIAL_OR_FIBROTIC_DISEASE_BOTH_
                      LUNGS
------ ------ 9 -- 0 OLD_AGE
------ ------ 3 -- 0 R_HILAR_ABNORMALITY
------ ------ 7 -- 0 DEMINERALIZED_BONES
------ ------ 5 -- 0 MALE

*** REPORT 1 ***
------ EFFECT 5 -- 5 INCR_LUCENCY_R_LUNG
CAUSE  EFFECT 3 -- 3 R_LUNG_TRIANGULAR_DENS
------ EFFECT 5 -- 4 POORLY_SEEN_RT_HEART_BORDER
------ EFFECT 5 -- 0 INCR_INTERSTIT_MARK_BOTH_LUNGS
CAUSE  ------ 1 -- 1 R_MID_LOBE_ATELECTASIS

POORLY_SEEN_RT_HEART_BORDER
     EXPLAINED BY
          R_LUNG_TRIANGULAR_DENS

INCR_LUCENCY_R_LUNG
AND R_LUNG_TRIANGULAR_DENS
AND INCR_INTERSTIT_MARK_BOTH_LUNGS
     EXPLAINED BY
          R_MID_LOBE_ATELECTASIS

*** REPORT 2 ***
------ EFFECT 5 -- 5 INCR_LUCENCY_R_LUNG
------ EFFECT 5 -- 5 CHRONIC_INTERSTITIAL_OR_FIBROTIC_DISEASE_BOTH_
                      LUNGS
CAUSE  EFFECT 3 -- 3 R_LUNG_TRIANGULAR_DENS
------ EFFECT 5 -- 4 POORLY_SEEN_RT_HEART_BORDER
CAUSE  ------ 1 -- 1 R_MID_LOBE_ATELECTASIS
CAUSE  ------ 6 -- 5 EMPHYSEMA_BOTH_LUNGS

INCR_LUCENCY_R_LUNG
AND CHRONIC_INTERSTITIAL_OR_FIBROTIC_DISEASE_BOTH_LUNGS
     EXPLAINED BY
          EMPHYSEMA_BOTH_LUNGS

POORLY_SEEN_RT_HEART_BORDER
     EXPLAINED BY
          R_LUNG_TRIANGULAR_DENS

R_LUNG_TRIANGULAR_DENS
     EXPLAINED BY
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R_MID_LOBE_ATELECTASIS

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First Year Subject RA8

*** 2 SEC ***
------ EFFECT 3 — 0 R_PERICARDIAL/HT_BORDER_INCR_DENS
CAUSE  ------ 3 — 3 R_HT_BORD_OR_PERICARD_MASS
CAUSE  ------ 0 — 0 R_HT_BORDER
------ EFFECT 5 — 6 INCR_LUCENCY_BOTH_LUNGS
CAUSE  ------ 5 — 5 CHRONIC_OBSTRUCTIVE_DISEASE_BOTH_LUNGS
------ EFFECT 5 — 0 BOTH_HEMIDIAPHRAGMS_FLAT
------ EFFECT 5 — 0 DECR_HT_SIZE

R_PERICARDIAL/HT_BORDER_INCR_DENS
     EXPLAINED BY
         R_HT_BORD_OR_PERICARD_MASS
          OR R_HT_BORDER

INCR_LUCENCY_BOTH_LUNGS
AND BOTH_HEMIDIAPHRAGMS_FLAT
AND DECR_HT_SIZE
     EXPLAINED BY
          CHRONIC_OBSTRUCTIVE_DISEASE_BOTH_LUNGS

*** PROMPT ***
------ ------ 3 — 0 R_PERICARDIAL/HT_BORDER_INCR_DENS
------ ------ 0 — 0 R_HT_BORDER
------ ------ 5 — 6 INCR_LUCENCY_BOTH_LUNGS
------ EFFECT 5 — 0 BOTH_HEMIDIAPHRAGMS_FLAT
CAUSE  ------ 5 — 5 CHRONIC_OBSTRUCTIVE_DISEASE_BOTH_LUNGS
------ ------ 3 — 0 PROM_R_HILUM
------ EFFECT 5 — 0 DECR_HT_SIZE

BOTH_HEMIDIAPHRAGMS_FLAT
AND DECR_HT_SIZE
     EXPLAINED BY
          CHRONIC_OBSTRUCTIVE_DISEASE_BOTH_LUNGS

*** REPORT 1 ***
------ EFFECT 3 — 5 INCR_DENS_R_MID_LUNG
------ EFFECT 4 — 0 INCR_DENS_R_LOWER_LUNG
------ EFFECT 5 — 5 INCR_LUCENCY_R_LUNG
CAUSE  EFFECT 5 — 5 R_LUNG_EMPHYSEMA
------ ------ 6 — 0 FLAT_R_DIAPHRAGM
CAUSE  ------ 0 — 0 R_HT_BORDER
CAUSE  ------ 6 — 0 R_LUNG_OBSTRUCTION

INCR_DENS_R_MID_LUNG
AND INCR_DENS_R_LOWER_LUNG
     EXPLAINED BY
          R_HT_BORDER

INCR_LUCENCY_R_LUNG
     EXPLAINED BY
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          R_LUNG_EMPHYSEMA

 R_LUNG_EMPHYSEMA
      EXPLAINED BY
           R_LUNG_OBSTRUCTION

*** REPORT 2 ***
------ EFFECT 3 — 5 INCR_DENS_R_MID_LUNG
CAUSE  ------ 0 — 0 R_HT_BORDER
CAUSE  ------ 0 — 4 R_PLEURAL_THICKENING
------ EFFECT 5 — 6 INCR_LUCENCY_BOTH_LUNGS
CAUSE  ------ 5 — 5 CHRONIC_OBSTRUCTIVE_DISEASE_BOTH_LUNGS

 INCR_DENS_R_MID_LUNG
      EXPLAINED BY
           R_HT_BORDER
           OR R_PLEURAL_THICKENING

 INCR_LUCENCY_BOTH_LUNGS
      EXPLAINED BY
           CHRONIC_OBSTRUCTIVE_DISEASE_BOTH_LUNGS
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C.B SECOND YEAR RESIDENTS

Second Year Subject RA9

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*** 2 SEC ***
------ ------ 5 — 0 INCR_LUCENCY_R_LOWER_LUNG
------ ------ 4 — 0 SOFT_TISSUE_DENS_INFERIOR_R_HT_BORDER

*** PROMPT ***
------ ------ 5 — 0 INCR_LUCENCY_R_LOWER_LUNG
------ ------ 4 — 0 SOFT_TISSUE_DENS_INFERIOR_R_HT_BORDER

*** REPORT 1 ***
------ ------ 5 — 4 POORLY_SEEN_RT_HEART_BORDER
------ ------ 3 — 3 SOFT_TISSUE_DENS_R_INFRA_HILUM
------ EFFECT 5 — 7 POORLY_SEEN_R_MEDIAL_DIAPHRAGM
------ EFFECT 5 — 5 INCR_LUCENCY_R_LUNG
CAUSE  ------ 1 — 1 R_MID_LOBE_ATELECTASIS
CAUSE  ------ 4 — 0 R_LUNG_PNEUMONIA
CAUSE  EFFECT 5 — 5 R_UPPER_LOBE_EXPANSION
CAUSE  ------ 3 — 0 R_LUNG_ATELECTASIS

POORLY_SEEN_R_MEDIAL_DIAPHRAGM
     EXPLAINED BY
          R_LUNG_PNEUMONIA
          OR R_LUNG_ATELECTASIS

INCR_LUCENCY_R_LUNG
     EXPLAINED BY
          R_UPPER_LOBE_EXPANSION

R_UPPER_LOBE_EXPANSION
     EXPLAINED BY
          R_MID_LOBE_ATELECTASIS

*** REPORT 2 ***
------ EFFECT 5 — 4 POORLY_SEEN_RT_HEART_BORDER
CAUSE  ------ 3 — 3 SOFT_TISSUE_DENS_R_INFRA_HILUM
------ ------ 5 — 7 POORLY_SEEN_R_MEDIAL_DIAPHRAGM
------ EFFECT 5 — 5 INCR_LUCENCY_R_LUNG
------ ------ 0 — 9 SOFT_TISSUE_NODULE_R_LATERAL_LUNG
------ EFFECT 7 — 9 INCR_VASC_L_LUNG
CAUSE  ------ 5 — 6 DECR_VASC_R_LUNG
CAUSE  ------ 1 — 1 R_MID_LOBE_ATELECTASIS
------ ------ 0 — 2 ATELECTASIS_R_LOWER_LOBE_MEDIALLY
------ ------ 0 — 2 PNEUMONIA_R_LOWER_LOBE_MEDIALLY
CAUSE  EFFECT 5 — 5 R_UPPER_LOBE_EXPANSION

POORLY_SEEN_RT_HEART_BORDER
     EXPLAINED BY
          SOFT_TISSUE_DENS_R_INFRA_HILUM
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INCR_VASC_L_LUNG
     EXPLAINED BY
          DECR_VASC_R_LUNG

INCR_LUCENCY_R_LUNG
     EXPLAINED BY
          R_UPPER_LOBE_EXPANSION

R_UPPER_LOBE_EXPANSION
     EXPLAINED BY
          R_MID_LOBE_ATELECTASIS
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Second Year Subject RA10

*** 2 SEC ***
------ ------ 2 -- 3 TRIANGULAR/WEDGED/SHARP_DENS_R_HT_BORD/PERICARDIAL

*** PROMPT ***
------ ------ 3 -- 0 R_PERICARDIAL/HT_BORDER_INCR_DENS

*** REPORT 1 ***
CAUSE  EFFECT 3 -- 0 R_PERICARDIAL/HT_BORDER_INCR_DENS
------ EFFECT 5 -- 4 POORLY_SEEN_RT_HEART_BORDER
CAUSE  ------ 0 -- 0 R_PARENCHYMAL_ORIGIN
CAUSE  ------ 0 -- 0 R_HILAR_ORIGIN
CAUSE  ------ 0 -- 0 CARDIAC_ORIGIN
------ EFFECT 5 -- 5 INCR_LUCENCY_R_LUNG
CAUSE  ------ 9 -- 0 ROTATION
------ EFFECT 7 -- 0 SPLAYED_VASC_R_LATERAL_LUNG
------ ------ 5 -- 5 CHRONIC_OBSTRUCTIVE_DISEASE_BOTH_LUNGS
------ ------ 9 -- 0 CALCIFIED_AORTIC_KNOB
------ EFFECT 9 -- 9 R_CLAVICLE_DEFORMITY
CAUSE  ------ 9 -- 9 HEALED_R_CLAVICLE_FRACTURE

POORLY_SEEN_RT_HEART_BORDER
     EXPLAINED_BY
          R_PERICARDIAL/HT_BORDER_INCR_DENS

R_PERICARDIAL/HT_BORDER_INCR_DENS
     EXPLAINED_BY
          R_PARENCHYMAL_ORIGIN
          OR R_HILAR_ORIGIN
          OR CARDIAC_ORIGIN

INCR_LUCENCY_R_LUNG
     EXPLAINED_BY
          ROTATION

SPLAYED_VASC_R_LATERAL_LUNG
     EXPLAINED_BY
          CHRONIC_OBSTRUCTIVE_DISEASE_R_LUNG

R_CLAVICLE_DEFORMITY
     EXPLAINED_BY
          HEALED_R_CLAVICLE_FRACTURE

*** REPORT 2 ***
CAUSE  EFFECT 3 -- 3 R_LUNG_TRIANGULAR_DENS
------ EFFECT 5 -- 4 POORLY_SEEN_RT_HEART_BORDER
------ ------ 0 -- 4 POORLY_SEEN_R_INFRA_HILUM
CAUSE  ------ 0 -- 0 CARDIAC_ORIGIN
CAUSE  ------ 0 -- 0 R_HILAR_ORIGIN
CAUSE  ------ 0 -- 0 ESOPHAGEAL_ORIGIN
CAUSE  ------ 0 -- 0 R_PARENCHYMAL_ORIGIN
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CAUSE  ------ 3 -- 3 R_HT_BORD_OR_PERICARD_MASS
CAUSE  ------ 4 -- 3 R_HILAR_MASS
CAUSE  ------ 0 -- 7 BRONCHOGENIC_CYST
------ EFFECT 5 -- 5 INCR_EXPANSION_R_LUNG
------ EFFECT 5 -- 6 DECR_VASC_R_LUNG
CAUSE  ------ 0 -- 5 CHRONIC_OBSTRUCTIVE_DISEASE_R_LUNG
------ EFFECT 9 -- 9 R_CLAVICLE_DEFORMITY
CAUSE  ------ 9 -- 9 HEALED_R_CLAVICLE_FRACTURE

POORLY_SEEN_RT_HEART_BORDER
AND POORLY_SEEN_R_HILUM
     EXPLAINED BY
          R_LUNG_TRIANGULAR_DENS

R_LUNG_TRIANGULAR_DENS
     EXPLAINED BY
          CARDIAC_ORIGIN
          OR R_HILAR_ORIGIN
          OR ESOPHAGEAL_ORIGIN
          OR R_PARENCHYMAL_ORIGIN
          OR R_HT_BORD_OR_PERICARD_MASS
          OR R_HILAR_MASS
          OR BRONCHOGENIC_CYST

INCR_EXPANSION_R_LUNG
AND DECR_VASC_R_LUNG
     EXPLAINED BY
          CHRONIC_OBSTRUCTIVE_DISEASE_R_LUNG

R_CLAVICLE_DEFORMITY
     EXPLAINED BY
          HEALED_R_CLAVICLE_FRACTURE
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Second Year Subject RA11

*** 2 SEC ***
------ ------ 7 — 9 ECTATIC_OR_TORTUOUS_OR_UNFOLDED_AORTA
------ ------ 3 — 0 INCR_DENS_R_LOWER_HT_BORDER
------ ------ 5 — 0 VERTICAL_LINEAR_DENS_R_UPPER_LUNG

*** PROMPT ***
------ ------ 5 — 0 INCR_DENS_R_LUNG_BASE
------ ------ 5 — 0 VERTICAL_LINEAR_DENS_R_UPPER_LUNG

*** REPORT 1 ***
------ EFFECT 3 — 3 SOFT_TISSUE_MASS_R_INFRA_HILUM
CAUSE  ------ 3 — 3 R_HT_BORD_OR_PERICARD_MASS
CAUSE  ------ 0 — 0 R_HILAR_ORIGIN
CAUSE  ------ 4 — 3 R_HILAR_MASS
CAUSE  ------ 3 — 3 R_PARASPINAL_MASS

SOFT_TISSUE_MASS_R_INFRA_HILUM
     EXPLAINED BY
          R_HT_BORD_OR_PERICARD_MASS
          OR R_HILAR_ORIGIN
          OR R_HILAR_MASS
          OR R_PARASPINAL_MASS

*** REPORT 2 ***
------ EFFECT 3 — 3 SOFT_TISSUE_MASS_R_INFRA_HILUM
CAUSE  ------ 0 — 0 R_HILAR_ORIGIN
CAUSE  ------ 3 — 3 R_PARASPINAL_MASS

SOFT_TISSUE_MASS_R_INFRA_HILUM
     EXPLAINED BY
          R_HILAR_ORIGIN
          OR R_PARASPINAL_MASS
```

C.C THIRD YEAR RESIDENTS

Third Year Subject RB1

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*** 2 SEC ***
------ ------ 3 — 3 R_LOWER_LUNG_SOFT_TISSUE_DENS
------ ------ 9 — 0 L_PERIHILAR_NODULE
------ ------ 9 — 0 INFLAMMATION_BOTH_LUNGS

*** PROMPT ***
------ ------ 9 — 0 NODULAR_DENS_L_PERIHILUM_SUPERIORLY
------ ------ 5 — 9 CHRONIC_POST-INFLAMMATION_BOTH_LUNGS
------ EFFECT 2 — 0 SOFT_TISSUE_DENS_R_LOWER_LUNG_MEDIALLY
CAUSE  ------ 5 — 0 CROWDED_VASC_R_LOWER_LUNG_MEDIALLY

SOFT_TISSUE_DENS_R_LOWER_LUNG_MEDIALLY
     EXPLAINED BY
          CROWDED_VASC_R_LOWER_LUNG_MEDIALLY

*** REPORT 1 ***
------ EFFECT 5 — 0 INCR_LUCENCY_R_UPPER_LUNG
------ EFFECT 5 — 0 STRINGY_OR_CURVILINEAR_DENS_R_UPPER_LUNG
CAUSE  ------ 5 — 5 BULLAE_R_UPPER_LUNG
------ EFFECT 9 — 9 RETICULAR_NODULAR_PATTERN_BOTH_LUNGS
------ EFFECT 3 — 0 MASS_R_LOWER_LUNG_MEDIALLY
CAUSE  ------ 4 — 0 CROWDED_R_HILAR_STRUCTURES
------ ------ 7 — 9 ECTATIC_OR_TORTUOUS_OR_UNFOLDED_AORTA
CAUSE  ------ 5 — 5 CHRONIC_OBSTRUCTIVE_DISEASE_BOTH_LUNGS

INCR_LUCENCY_R_UPPER_LUNG
AND STRINGY_OR_CURVILINEAR_DENS_R_UPPER_LUNG
     EXPLAINED BY
          BULLAE_R_UPPER_LUNG

MASS_R_LOWER_LUNG_MEDIALLY
     EXPLAINED BY
          CROWDED_R_HILAR_STRUCTURES

RETICULAR_NODULAR_PATTERN_BOTH_LUNGS
AND MASS_R_LOWER_LUNG_MEDIALLY
     EXPLAINED BY
          CHRONIC_OBSTRUCTIVE_DISEASE_BOTH_LUNGS

*** REPORT 2 ***
------ EFFECT 0 — 5 INCR_LATERAL_TO_LATERAL_THORAX
------ EFFECT 0 — 5 INCR_SUPERIOR_TO_INFERIOR_THORAX
------ ------ 5 — 5 BULLAE_R_UPPER_LUNG
------ EFFECT 9 — 9 RETICULAR_NODULAR_PATTERN_BOTH_LUNGS
------ EFFECT 0 — 4 CROWDED_R_HILAR_VESSELS
------ ------ 7 — 9 ECTATIC_OR_TORTUOUS_OR_UNFOLDED_AORTA
CAUSE  ------ 5 — 5 CHRONIC_OBSTRUCTIVE_DISEASE_BOTH_LUNGS
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INCR_SUPERIOR_TO_INFERIOR_THORAX
AND INCR_LATERAL_TO_LATERAL_THORAX
AND RETICULAR_NODULAR_PATTERN_BOTH_LUNGS
AND CROWDED_R_HILAR_VESSELS
     EXPLAINED BY
          CHRONIC_OBSTRUCTIVE_DISEASE_BOTH_LUNGS
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Third Year Subject RB2

*** 2 SEC ***
------ ------ 3 — 3 R_PARASPINAL_MASS

*** PROMPT ***
CAUSE  ------ 3 — 3 SHARP_R_PARASPINAL_MASS
------ EFFECT 5 — 4 POORLY_SEEN_RT_HEART_BORDER

POORLY_SEEN_RT_HEART_BORDER
     EXPLAINED BY
          SHARP_R_PARASPINAL_MASS

*** REPORT 1 ***
------ EFFECT 3 — 3 R_PARASPINAL_MASS
CAUSE  ------ 3 — 0 NEOPLASM_R_PARASPINAL_AT_HT_LEVEL

R_PARASPINAL_MASS
     EXPLAINED BY
          NEOPLASM_R_PARASPINAL_AT_HT_LEVEL

*** REPORT 2 ***
------ EFFECT 3 — 3 R_PARASPINAL_MASS
CAUSE  ------ 0 — 3 R_PARASPINAL_BENIGN_TUMOR
CAUSE  ------ 0 — 3 BRONCHOPULMONARY_SEQUESTRATION
CAUSE  ------ 0 — 3 R_PARASPINAL_BRONCHOGENIC_CYST

R_PARASPINAL_MASS
     EXPLAINED BY
          R_PARASPINAL_BENIGN_TUMOR
          OR BRONCHOPULMONARY_SEQUESTRATION
          OR R_PARASPINAL_BRONCHOGENIC_CYST
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Third Year Subject RB3

*** 2 SEC ***
CAUSE  EFFECT 3 — 0 R_MIDDLE_MEDIASTINAL_MASS
------ EFFECT 5 — 4 POORLY_SEEN_RT_HEART_BORDER
------ ------ 5 — 0 MIDDLE_AGE
------ ------ 5 — 0 INCR_INTERSTIT_MARK_BOTH_LUNGS
CAUSE  ------ 1 — 1 R_MID_LOBE_ATELECTASIS

POORLY_SEEN_RT_HEART_BORDER
     EXPLAINED BY
          R_MIDDLE_MEDIASTINAL_MASS

R_MIDDLE_MEDIASTINAL_MASS
     EXPLAINED BY
          R_MID_LOBE_ATELECTASIS

*** PROMPT ***
------ EFFECT 3 — 3 R_HT_BORD_OR_PERICARD_MASS
CAUSE  ------ 1 — 1 R_MID_LOBE_ATELECTASIS
------ ------ 5 — 0 INCR_INTERSTIT_MARK_BOTH_LUNGS
------ ------ 3 — 0 ABNORMAL_R_HEART_CONFIGURATION

R_HT_BORD_OR_PERICARD_MASS
     EXPLAINED BY
          R_MID_LOBE_ATELECTASIS

*** REPORT 1 ***
CAUSE  EFFECT 3 — 0 R_PERICARDIAL/HT_BORDER_INCR_DENS
------ EFFECT 3 — 0 ABNORMAL_R_HEART_CONFIGURATION
------ EFFECT 5 — 4 POORLY_SEEN_RT_HEART_BORDER
CAUSE  ------ 3 — 3 R_HT_BORD_OR_PERICARD_MASS
CAUSE  ------ 1 — 1 R_MID_LOBE_ATELECTASIS

ABNORMAL_R_HEART_CONFIGURATION
OR POORLY_SEEN_RT_HEART_BORDER
     EXPLAINED BY
          R_PERICARDIAL/HT_BORDER_INCR_DENS

R_PERICARDIAL/HT_BORDER_INCR_DENS
     EXPLAINED BY
          R_HT_BORD_OR_PERICARD_MASS
          OR R_MID_LOBE_ATELECTASIS

*** REPORT 2 ***
------ EFFECT 0 — 9 ENLARGED_ESOPHAGUS_WITH_AIR_FLUID_LEVEL
CAUSE  ------ 0 — 9 ESOPHAGEAL_ACHALASIA
CAUSE  ------ 0 — 9 DISTAL_ESOPHAGEAL_STRICTURE
CAUSE  ------ 0 — 9 ESOPHAGEAL_CARCINOMA

ENLARGED_ESOPHAGUS_WITH_AIR_FLUID_LEVEL
     EXPLAINED BY
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ESOPHAGEAL_ACHALASIA
OR DISTAL_ESOPHAGEAL_STRICTURE
OR ESOPHAGEAL_CARCINOMA

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Third Year Subject RB4

*** 2 SEC ***
------ ------ 3 -- 0 R_PERICARDIAL/HT_BORDER_INCR_DENS

*** PROMPT ***
------ EFFECT 3 -- 0 R_PERICARDIAL/HT_BORDER_INCR_DENS
CAUSE  ------ 0 -- 0 GREAT_VESSELS
CAUSE  ------ 0 -- 0 CARDIAC_ORIGIN

R_PERICARDIAL/HT_BORDER_INCR_DENS
     EXPLAINED BY
          GREAT_VESSELS
          OR CARDIAC_ORIGIN

*** REPORT 1 ***
------ EFFECT 4 -- 0 INCR_DENS_R_HILUM
CAUSE  ------ 0 -- 0 VASCULATURE

INCR_DENS_R_HILUM
     EXPLAINED BY
          VASCULATURE

*** REPORT 2 ***
------ ------ 0 -- 9 ECTATIC_VASC_R_LUNG
------ ------ 5 -- 5 EMPHYSEMA_R_UPPER_LOBE
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C.D FOURTH YEAR RESIDENTS

Fourth Year Subject RB5

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*** 2 SEC ***
------ EFFECT 3 -- 3 R_LOWER_LUNG_SOFT_TISSUE_DENS
CAUSE  ------ 0 -- 0 R_LUNG_ORIGIN
CAUSE  ------ 0 -- 0 PLEURAL_ORIGIN
CAUSE  ------ 0 -- 0 ESOPHAGEAL_ORIGIN
CAUSE  ------ 7 -- 9 ECTATIC_OR_TORTUOUS_OR_UNFOLDED_AORTA
------ ------ 9 -- 0 OLD_AGE

R_LOWER_LUNG_SOFT_TISSUE_DENS
     EXPLAINED BY
          R_LUNG_ORIGIN
          OR PLEURAL_ORIGIN
          OR ECTATIC_OR_TORTUOUS_OR_UNFOLDED_AORTA
          OR ESOPHAGEAL_ORIGIN

*** PROMPT ***
------ ------ 1 -- 0 SOFT_TISSUE_DENS_MEDIAL_R_LOWER_LOBE
------ ------ 5 -- 5 CHRONIC_INTERSTITIAL_OR_FIBROTIC_DISEASE_BOTH_
                       LUNGS
------ ------ 6 -- 0 PROM_VASC_BOTH_LUNGS
------ ------ 5 -- 6 INCR_LUCENCY_BOTH_LUNGS
------ EFFECT 0 -- 0 SKELETAL_STRUCTURES
CAUSE  ------ 0 -- 0 NORMAL_IF_AGE_ADJUSTED

SKELETAL_STRUCTURES
     EXPLAINED BY
          NORMAL_IF_AGE_ADJUSTED

*** REPORT 1 ***
------ ------ 5 -- 0 MIDDLE_AGE
------ ------ 5 -- 0 MALE
------ ------ 3 -- 3 SOFT_TISSUE_DENS_R_INFRA_HILUM
------ ------ 3 -- 3 SOFT_TISSUE_MASS_R_INFRA_HILUM
CAUSE  ------ 5 -- 0 STRINGY_DENS_R_LUNG
------ EFFECT 5 -- 7 POORLY_SEEN_R_MEDIAL_DIAPHRAGM
------ EFFECT 3 -- 0 POORLY_SEEN_R_HILUM
CAUSE  ------ 3 -- 0 R_HILAR_ADENOPATHY
------ EFFECT 8 -- 9 UNUSUAL_OR_SPLAYED_OR_PROM_OR_CROWDED_VASC_R_LUNG
CAUSE  ------ 5 -- 5 CHRONIC_INTERSTITIAL_OR_FIBROTIC_DISEASE_R_LUNG
------ ------ 3 -- 0 BRONCHOGENIC_CARCINOMA_R_LOWER_LOBE

POORLY_SEEN_R_MEDIAL_DIAPHRAGM
     EXPLAINED BY
          STRINGY_DENS_R_LUNG

UNUSUAL_OR_SPLAYED_OR_PROM_OR_CROWDED_VASC_R_LUNG
     EXPLAINED BY
          CHRONIC_INTERSTITIAL_OR_FIBROTIC_DISEASE_R_LUNG
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POORLY_SEEN_R_HILUM
     EXPLAINED BY
          R_HILAR_ADENOPATHY

*** REPORT 2 ***
------ EFFECT 3 — 3 SOFT_TISSUE_MASS_R_INFRA_HILUM
------ ------ 5 — 7 POORLY_SEEN_R_MEDIAL_DIAPHRAGM
CAUSE  ------ 0 — 9 ACTIVE_DISEASE_BOTH_LUNGS
CAUSE  ------ 5 — 9 CHRONIC_POST-INFLAMMATION_BOTH_LUNGS

SOFT_TISSUE_MASS_R_INFRA_HILUM
     EXPLAINED BY
          ACTIVE_DISEASE_BOTH_LUNGS
          OR CHRONIC_POST-INFLAMMATION_BOTH_LUNGS
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Fourth Year Subject RB6

*** 2 SEC ***
------ EFFECT 2 — 0 R_MID_LUNG_HT_BORD_OR_PARASPINAL_TRIANGULAR_DENS
CAUSE  ------ 1 — 0 R_MID_LUNG_ABNORMALITY
------ EFFECT 5 — 4 POORLY_SEEN_RT_HEART_BORDER
CAUSE  ------ 9 — 9 ABNORMAL_HEART_CONFIGURATION
------ ------ 5 — 0 INCR_LUCENCY_R_UPPER_LOBE
------ ------ 5 — 5 CHRONIC_INTERSTITIAL_OR_FIBROTIC_DISEASE_BOTH
                       LUNGS
------ ------ 7 — 0 BULLAE_OR_BLEBS_BOTH_LUNGS

R_MID_LUNG_HT_BORD_OR_PARASPINAL_TRIANGULAR_DENS
     EXPLAINED BY
          ABNORMAL_HEART_CONFIGURATION
          OR R_MID_LUNG_ABNORMALITY

POORLY_SEEN_RT_HEART_BORDER
     EXPLAINED BY
          R_MID_LUNG_ABNORMALITY

*** PROMPT ***
------ ------ 5 — 0 INCR_EXPANSION_BOTH_LUNGS
------ EFFECT 5 — 0 INCR_LUCENCY_R_UPPER_LUNG
CAUSE  EFFECT 5 — 5 BULLAE_R_UPPER_LUNG
CAUSE  ------ 5 — 0 R_UPPER_LUNG_EMPHYSEMA
------ ------ 5 — 0 BOTH_HEMIDIAPHRAGMS_FLAT
------ EFFECT 3 — 0 ABNORMAL_R_HEART_CONFIGURATION
CAUSE  ------ 3 — 0 R_MEDIASTINAL_MASS
CAUSE  ------ 3 — 0 LUNG_MASS_R_HT_AREA

INCR_LUCENCY_R_UPPER_LUNG
     EXPLAINED BY
          BULLAE_R_UPPER_LUNG

BULLAE_R_UPPER_LUNG
     EXPLAINED BY
          R_UPPER_LUNG_EMPHYSEMA

ABNORMAL_R_HEART_CONFIGURATION
     EXPLAINED BY
          LUNG_MASS_R_HT_AREA
          OR R_MEDIASTINAL_MASS
          OR R_CARDIAC_MASS

*** REPORT 1 ***
------ EFFECT 5 — 4 POORLY_SEEN_RT_HEART_BORDER
CAUSE  EFFECT 0 — 0 R_HT_BORD_OR_PERICARD_OR_PARASPINAL_TRIANGULAR_
                      OR_WEDGED_OR_SHARP_DENS
------ EFFECT 5 — 5 INCR_EXPANSION_R_LUNG
------ EFFECT 5 — 5 INCR_LUCENCY_R_LUNG
CAUSE  ------ 5 — 6 DECR_VASC_R_LUNG
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CAUSE  ------ 5 -- 0 DECR_VASC_R_LUNG_APEX
CAUSE  ------ 5 -- 5 R_LUNG_EMPHYSEMA
------ ------ 5 -- 5 BULLAE_R_LUNG_APEX
------ ------ 5 -- 5 BULLAE_LATERAL_R_MID_LUNG
CAUSE  ------ 1 -- 1 R_MID_LOBE_ATELECTASIS

INCR_LUCENCY_R_LUNG
AND INCR_EXPANSION_R_LUNG
     EXPLAINED BY
          DECR_VASC_R_LUNG
          AND DECR_VASC_R_LUNG_APEX
          AND R_LUNG_EMPHYSEMA

POORLY_SEEN_RT_HEART_BORDER
     EXPLAINED BY
          R_HT_BORD_OR_PERICARD_OR_PARASPINAL_TRIANGULAR_OR_WEDGED_
            OR_SHARP_DENS

R_HT_BORD_OR_PERICARD_OR_PARASPINAL_TRIANGULAR_OR_WEDGED_OR_SHARP_DENS
     EXPLAINED BY
          R_MID_LOBE_ATELECTASIS

*** REPORT 2 ***
------ ------ 5 -- 5 INCR_EXPANSION_R_LUNG
------ ------ 5 -- 5 INCR_LUCENCY_R_LUNG
------ ------ 5 -- 5 BULLAE_R_LUNG_APEX
------ ------ 5 -- 5 BULLAE_LATERAL_R_MID_LUNG
------ EFFECT 2 -- 3 TRIANGULAR/WEDGED/SHARP_DENS_R_HT_BORD/PERICARDIAL
CAUSE  ------ 1 -- 1 R_MID_LOBE_ATELECTASIS

TRIANGULAR/WEDGED/SHARP_DENS_R_HT_BORD/PERICARDIAL
     EXPLAINED BY
          R_MID_LOBE_ATELECTASIS
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Fourth Year Subject RB7

*** 2 SEC ***
------ EFFECT 2 -- 3 TRIANGULAR/WEDGED/SHARP_DENS_R_HT_BORD/PERICARDIAL
------ EFFECT 5 -- 4 POORLY_SEEN_RT_HEART_BORDER
CAUSE  ------ 1 -- 0 R_MID_LOBE_LESION
CAUSE  ------ 9 -- 9 ABNORMAL_HEART_CONFIGURATION
------ EFFECT 5 -- 0 INCR_EXPANSION_BOTH_LUNGS
CAUSE  ------ 6 -- 5 EMPHYSEMA_BOTH_LUNGS

INCR_EXPANSION_BOTH_LUNGS
     EXPLAINED BY
          EMPHYSEMA_BOTH_LUNGS

POORLY_SEEN_RT_HEART_BORDER
     EXPLAINED BY
          R_MID_LOBE_LESION

TRIANGULAR/WEDGED/SHARP_DENS_R_HT_BORD/PERICARDIAL
     EXPLAINED BY
          R_MID_LOBE_LESION
          OR ABNORMAL_HEART_CONFIGURATION

*** PROMPT ***
------ EFFECT 3 -- 0 ABNORMAL_R_INFRA_HILUM
CAUSE  ------ 3 -- 0 R_HILAR_LESION
CAUSE  ------ 3 -- 0 LUNG_LESION_NEAR_R_HT_BORDER
CAUSE  ------ 3 -- 0 R_CARDIAC_LESION
------ EFFECT 5 -- 0 CYSTIC_CHANGE_BOTH_LUNGS
CAUSE  ------ 6 -- 5 EMPHYSEMA_BOTH_LUNGS
------ EFFECT 5 -- 0 BOTH_HEMIDIAPHRAGMS_FLAT
CAUSE  ------ 5 -- 0 INCR_EXPANSION_BOTH_LUNGS
CAUSE  ------ 3 -- 3 R_HT_BORD_OR_PERICARD_MASS
CAUSE  ------ 3 -- 0 R_HT_BORD_INFILTRATE
------ ------ 9 -- 0 INCR_HT_SIZE

BOTH_HEMIDIAPHRAGMS_FLAT
     EXPLAINED BY
          INCR_EXPANSION_BOTH_LUNGS

CYSTIC_CHANGE_BOTH_LUNGS
     EXPLAINED BY
          EMPHYSEMA_BOTH_LUNGS

ABNORMAL_R_INFRA_HILUM
     EXPLAINED BY
          LUNG_LESION_NEAR_R_HT_BORDER
          OR R_HILAR_LESION
          OR R_CARDIAC_LESION
          OR R_HT_BORD_INFILTRATE
          OR R_HT_BORD_OR_PERICARD_MASS
          OR R_HT_ENLARGEMENT
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*** REPORT 1 ***
------ ------ 5 -- 5 R_LUNG_EMPHYSEMA
------ EFFECT 3 -- 3 R_LOWER_LUNG_SOFT_TISSUE_DENS
CAUSE  ------ 4 -- 0 R_LOWER_LUNG_ATELECTASIS
CAUSE  ------ 4 -- 3 R_HILAR_MASS
CAUSE  ------ 8 -- 5 UNUSUAL_OR_SPLAYED_OR_PROM_OR_CROWDED_VASC_R_
                       LOWER_LUNG

R_LOWER_LUNG_SOFT_TISSUE_DENS
     EXPLAINED BY
          R_HILAR_MASS
          OR R_LOWER_LUNG_ATELECTASIS
          OR UNUSUAL_OR_SPLAYED_OR_PROM_OR_CROWDED_VASC_R_LOWER_LUNG

*** REPORT 2 ***
------ ------ 6 -- 5 EMPHYSEMA_BOTH_LUNGS
------ ------ 5 -- 5 R_LUNG_EMPHYSEMA
------ EFFECT 3 -- 3 R_LOWER_LUNG_SOFT_TISSUE_DENS
CAUSE  ------ 8 -- 5 UNUSUAL_OR_SPLAYED_OR_PROM_OR_CROWDED_VASC_R_
                       LOWER_LUNG

R_LOWER_LUNG_SOFT_TISSUE_DENS
     EXPLAINED BY
          UNUSUAL_OR_SPLAYED_OR_PROM_OR_CROWDED_VASC_R_LOWER_LUNG
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C.E EXPERTS

Expert Subject E1

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*** 2 SEC ***
------ EFFECT 0 — 0 MEDIASTINUM_R_TO_L_SHIFT
CAUSE  ------ 5 — 5 INCR_EXPANSION_R_LUNG
------ ------ 5 — 5 INCR_LUCENCY_R_LUNG
------ EFFECT 0 — 0 PROM_OR_COMPRESSED_OR_CROWDED_VESSELS_LEFT_LOWER_
                      LOBE
CAUSE  ------ 0 — 0 HEART_SHIFTED_R_TO_L
------ ------ 5 — 0 EMPHYSEMA_R_LOWER_LOBE
------ ------ 0 — 0 EMPHYSEMA_RT_MID_LOBE

MEDIASTINUM_R_TO_L_SHIFT
     EXPLAINED BY
          INCR_EXPANSION_R_LUNG

PROM_OR_COMPRESSED_OR_CROWDED_VESSELS_LEFT_LOWER_LOBE
     EXPLAINED BY
          HEART_SHIFTED_R_TO_L

*** PROMPT ***
------ EFFECT 5 — 5 INCR_EXPANSION_R_LUNG
------ EFFECT 5 — 5 INCR_LUCENCY_R_LUNG
------ EFFECT 0 — 0 INCR_EXPANSION_RT_MIDDLE_LOBE
------ EFFECT 0 — 0 INCR_LUCENCY_RT_MIDDLE_LOBE
------ EFFECT 0 — 0 INCR_EXPANSION_RT_LOWER_LOBE
------ EFFECT 0 — 0 INCR_LUCENCY_RT_LOWER_LOBE
CAUSE  ------ 5 — 5 R_LUNG_EMPHYSEMA
------ EFFECT 0 — 0 SMALL_RT_HILUM/HILAR_VESSELS
------ EFFECT 0 — 0 LARGE_LEFT_HILUM/HILAR_VESSELS
CAUSE  EFFECT 0 — 0 INCR_BLOOD_FLOW_L_LUNG
------ ------ 0 — 0 R_DIAPHRAGM_ELEVATION
------ ------ 6 — 0 FLAT_R_DIAPHRAGM
------ ------ 0 — 0 MEDIASTINUM_R_TO_L_SHIFT
------ ------ 0 — 0 HEART_SHIFTED_R_TO_L

INCR_EXPANSION_R_LUNG
AND INCR_LUCENCY_R_LUNG
AND INCR_EXPANSION_RT_MIDDLE_LOBE
AND INCR_LUCENCY_RT_MIDDLE_LOBE
AND INCR_EXPANSION_RT_LOWER_LOBE
AND INCR_LUCENCY_RT_LOWER_LOBE
AND SMALL_RT_HILUM/HILAR_VESSELS
AND INCR_BLOOD_FLOW_L_LUNG
     EXPLAINED BY
          R_LUNG_EMPHYSEMA

LARGE_LEFT_HILUM/HILAR_VESSELS
     EXPLAINED BY
          INCR_BLOOD_FLOW_L_LUNG
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*** REPORT 1 ***
------ ------ 5 — 5 INCR_EXPANSION_R_LUNG
------ EFFECT 0 — 0 LINEAR_MARKINGS_RT_LUNG
CAUSE  EFFECT 5 — 5 R_LUNG_EMPHYSEMA
------ EFFECT 0 — 0 SMALL_PULMONARY_ARTERIES_RT_LUNG
------ EFFECT 0 — 0 STRETCHED_PULMONARY_ARTERIES_RT_LUNG
------ EFFECT 2 — 3 TRIANGULAR/WEDGED/SHARP_DENS_R_HT_BORD/PERICARDIAL
CAUSE  EFFECT 0 — 0 ATELECTASIS_SUPERIOR_SEG_R_LOWER_LOBE
CAUSE  EFFECT 0 — 2 ATELECTASIS_R_LOWER_LOBE_MEDIALLY
------ EFFECT 0 — 0 ROUND_DENS_RT_5TH_RIB_AREA
CAUSE  EFFECT 0 — 0 MALIGNANCY_RT_5TH_RIB_AREA
CAUSE  EFFECT 0 — 0 METASTASIS_RT_5TH_RIB_AREA
CAUSE  ------ 0 — 0 GRANULOMA_RT_5TH_RIB_AREA
CAUSE  ------ 0 — 0 HIDDEN_MASS_R_HILUS
CAUSE  EFFECT 0 — 0 OBSTRUCTION_L_MAIN_STEM_BRONCHUS

LINEAR_MARKINGS_RT_LUNG
AND SMALL_PULMONARY_ARTERIES_RT_LUNG
AND STRETCHED_PULMONARY_ARTERIES_RT_LUNG
     EXPLAINED BY
          R_LUNG_EMPHYSEMA

TRIANGULAR/WEDGED/SHARP_DENS_R_HT_BORD/PERICARDIAL
     EXPLAINED BY
          ATELECTASIS_SUPERIOR_SEG_R_LOWER_LOBE
          OR ATELECTASIS_R_LOWER_LOBE_MEDIALLY

ATELECTASIS_SUPERIOR_SEG_R_LOWER_LOBE
OR ATELECTASIS_R_LOWER_LOBE_MEDIALLY
     EXPLAINED BY
          OBSTRUCTION_L_MAIN_STEM_BRONCHUS

ROUND_DENS_RT_5TH_RIB_AREA
     EXPLAINED BY
          MALIGNANCY_RT_5TH_RIB_AREA
          OR GRANULOMA_RT_5TH_RIB_AREA

MALIGNANCY_RT_5TH_RIB_AREA
     EXPLAINED BY
          METASTASIS_RT_5TH_RIB_AREA

R_LUNG_EMPHYSEMA
     EXPLAINED BY
          OBSTRUCTION_L_MAIN_STEM_BRONCHUS

METASTASIS_RT_5TH_RIB_AREA
AND OBSTRUCTION_L_MAIN_STEM_BRONCHUS
     EXPLAINED BY
          HIDDEN_MASS_R_HILUS

*** REPORT 2 ***
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CAUSE  EFFECT 0 — 0 HIDDEN_MEDIASTINAL_MASS
CAUSE  EFFECT 0 — 0 HIDDEN_METASTASIS_MEDIASTINUM
CAUSE  ------ 0 — 0 MALIGNANCY_OF_GASTROINTESTINAL_TRACT
------ EFFECT 5 — 5 R_LUNG_EMPHYSEMA
------ EFFECT 0 — 0 METASTASIS_RT_5TH_RIB_AREA

HIDDEN_MEDIASTINAL_MASS
     EXPLAINED BY
          HIDDEN_METASTASIS_MEDIASTINUM

HIDDEN_METASTASIS_MEDIASTINUM
     EXPLAINED BY
          MALIGNANCY_OF_GASTROINTESTINAL_TRACT

R_LUNG_EMPHYSEMA
AND METASTASIS_RT_5TH_RIB_AREA
     EXPLAINED BY
          HIDDEN_MEDIASTINAL_MASS
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Expert Subject E2

*** 2 SEC ***
CAUSE  EFFECT 0 — 0 R_HT_BORD_OR_PERICARD_OR_PARASPINAL_TRIANGULAR_
                     OR_WEDGED_OR_SHARP_DENS
------ EFFECT 5 — 4 POORLY_SEEN_RT_HEART_BORDER
CAUSE  EFFECT 1 — 0 R_MID_LOBE_LESION
CAUSE  ------ 0 — 0 R_MIDDLE_LOBE_PNEUMONIA
------ ------ 6 — 5 EMPHYSEMA_BOTH_LUNGS
CAUSE  ------ 5 — 5 CHRONIC_OBSTRUCTIVE_DISEASE_BOTH_LUNGS
------ EFFECT 0 — 0 STRINGY_OR_WAVY_DENSITY_R_UPPER_LOBE
CAUSE  ------ 5 — 5 BULLAE_R_UPPER_LOBE
------ EFFECT 0 — 0 HYPOPLASTIC_HEART

POORLY_SEEN_RT_HEART_BORDER
     EXPLAINED BY
          R_HT_BORD_OR_PERICARD_OR_PARASPINAL_TRIANGULAR_OR_WEDGED_
           OR_SHARP_DENS

R_HT_BORD_OR_PERICARD_OR_PARASPINAL_TRIANGULAR_OR_WEDGED_OR_SHARP_DENS
AND POORLY_SEEN_RT_HEART_BORDER
     EXPLAINED BY
          R_MID_LOBE_LESION

R_HT_BORD_OR_PERICARD_OR_PARASPINAL_TRIANGULAR_OR_WEDGED_OR_SHARP_DENS
AND POORLY_SEEN_RT_HEART_BORDER
AND R_MID_LOBE_LESION
     EXPLAINED BY
          R_MIDDLE_LOBE_PNEUMONIA

STRINGY_OR_WAVY_DENSITY_R_UPPER_LOBE
     EXPLAINED BY
          BULLAE_R_UPPER_LOBE

HYPOPLASTIC_HEART
     EXPLAINED BY
          CHRONIC_OBSTRUCTIVE_DISEASE_BOTH_LUNGS

*** PROMPT ***
------ ------ 5 — 6 INCR_LUCENCY_BOTH_LUNGS
------ EFFECT 0 — 0 STRINGY_OR_WAVY_DENSITY_R_UPPER_LOBE
CAUSE  ------ 5 — 5 BULLAE_R_UPPER_LOBE
CAUSE  EFFECT 0 — 0 R_HT_BORD_OR_PERICARD_OR_PARASPINAL_TRIANGULAR_
                     OR_WEDGED_OR_SHARP_DENS
------ EFFECT 5 — 4 POORLY_SEEN_RT_HEART_BORDER
CAUSE  ------ 0 — 0 R_MIDDLE_LOBE_PNEUMONIA
CAUSE  ------ 1 — 1 R_MID_LOBE_ATELECTASIS
------ EFFECT 5 — 0 BOTH_HEMIDIAPHRAGMS_FLAT
CAUSE  ------ 5 — 0 INCR_EXPANSION_BOTH_LUNGS
CAUSE  ------ 5 — 5 CHRONIC_OBSTRUCTIVE_DISEASE_BOTH_LUNGS
------ EFFECT 0 — 0 NARROW_HEART
------ EFFECT 0 — 0 HYPOPLASTIC_HEART
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CAUSE  ------ 0 — 0 AIR_TRAPPING_BOTH_LUNGS
------ ------ 0 — 0 OSTEOPENIC_BONES

STRINGY_OR_WAVY_DENSITY_R_UPPER_LOBE
     EXPLAINED BY
          BULLAE_R_UPPER_LOBE

POORLY_SEEN_RT_HEART_BORDER
     EXPLAINED BY
          R_HT_BORD_OR_PERICARD_OR_PARASPINAL_TRIANGULAR_OR_WEDGED_
            OR_SHARP_DENS

R_HT_BORD_OR_PERICARD_OR_PARASPINAL_TRIANGULAR_OR_WEDGED_OR_SHARP_DENS
     EXPLAINED BY
          R_MIDDLE_LOBE_PNEUMONIA
          OR R_MID_LOBE_ATELECTASIS

BOTH_HEMIDIAPHRAGMS_FLAT
     EXPLAINED BY
          INCR_EXPANSION_BOTH_LUNGS
          AND CHRONIC_OBSTRUCTIVE_DISEASE_BOTH_LUNGS

NARROW_HEART
AND HYPOPLASTIC_HEART
     EXPLAINED BY
          CHRONIC_OBSTRUCTIVE_DISEASE_BOTH_LUNGS
          OR AIR_TRAPPING_BOTH_LUNGS

*** REPORT 1 ***
------ ------ 5 — 5 CHRONIC_OBSTRUCTIVE_DISEASE_BOTH_LUNGS
CAUSE  ------ 0 — 0 CHRONIC_BULLOUS_EMPHYSEMA_BOTH_LUNGS
------ EFFECT 0 — 0 R_HT_BORD_OR_PERICARD_OR_PARASPINAL_TRIANGULAR_
                      OR_WEDGED_OR_SHARP_DENS
CAUSE  EFFECT 1 — 1 R_MID_LOBE_ATELECTASIS
CAUSE  ------ 0 — 0 NEOPLASM
CAUSE  ------ 0 — 0 MUCUS_PLUGGING

R_HT_BORD_OR_PERICARD_OR_PARASPINAL_TRIANGULAR_OR_WEDGED_OR_SHARP_DENS
     EXPLAINED BY
          R_MID_LOBE_ATELECTASIS

R_MID_LOBE_ATELECTASIS
     EXPLAINED BY
          CHRONIC_BULLOUS_EMPHYSEMA_BOTH_LUNGS
          OR NEOPLASM
          OR MUCUS_PLUGGING

*** REPORT 2 ***
CAUSE  ------ 0 — 0 CHRONIC_BULLOUS_EMPHYSEMA_BOTH_LUNGS
------ EFFECT 0 — 0 R_HT_BORD_OR_PERICARD_OR_PARASPINAL_TRIANGULAR_
                      OR_WEDGED_OR_SHARP_DENS
CAUSE  ------ 1 — 1 R_MID_LOBE_ATELECTASIS
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```
------ EFFECT 0 — 0 HYPOPLASTIC_HEART
------ EFFECT 7 — 9 ECTATIC_OR_TORTUOUS_OR_UNFOLDED_AORTA
CAUSE  ------ 0 — 0 NORMAL_IF_AGE_ADJUSTED
------ EFFECT 0 — 0 CALCIFIED_AORTA
------ EFFECT 0 — 0 COIN_LESION_4TH_INTERSPACE_RT
CAUSE  ------ 0 — 0 CALCIFIED_DENSITY_4TH_INTERSPACE_RT
CAUSE  ------ 0 — 0 MALIGNANT_LESION_4TH_INTERSPACE_RT

R_HT_BORD_OR_PERICARD_OR_PARASPINAL_TRIANGULAR_OR_WEDGED_OR_SHARP_DENS
     EXPLAINED BY
          R_MID_LOBE_ATELECTASIS

HYPOPLASTIC_HEART
     EXPLAINED BY
          CHRONIC_BULLOUS_EMPHYSEMA_BOTH_LUNGS

ECTATIC_OR_TORTUOUS_OR_UNFOLDED_AORTA
AND CALCIFIED_AORTA
     EXPLAINED BY
          NORMAL_IF_AGE_ADJUSTED

COIN_LESION_4TH_INTERSPACE_RT
     EXPLAINED BY
          CALCIFIED_DENSITY_4TH_INTERSPACE_RT
          OR MALIGNANT_LESION_4TH_INTERSPACE_RT
```

```
Expert Subject E3

*** 2 SEC ***
------ ------ 0 — 0 R_HT_BORD_OR_PERICARD_AND_OR_PARASPINAL_DENS_
                      OR_BULGE_OR_PROM_OR_SUSPECT_AREA
------ ------ 0 — 0 INCR_DENS_BEHIND_HT
------ ------ 5 — 6 INCR_LUCENCY_BOTH_LUNGS

*** PROMPT ***
------ ------ 5 — 6 INCR_LUCENCY_BOTH_LUNGS
------ ------ 3 — 0 INCR_DENS_R_LOWER_HT_BORDER
------ ------ 0 — 0 INCR_DENS_BEHIND_HT

*** REPORT 1 ***
------ ------ 5 — 6 INCR_LUCENCY_BOTH_LUNGS
------ EFFECT 5 — 5 INCR_LUCENCY_R_LUNG
CAUSE  ------ 0 — 0 IDIOPATHIC_HYPERLUCENT_LUNG_RT_SIDE
------ ------ 3 — 0 INCR_DENS_R_LOWER_HT_BORDER
------ ------ 0 — 0 NODULAR_DENSITY_RT_LUNG_ADJACENT_TO_SCAPULAR_ANGLE

INCR_LUCENCY_R_LUNG
     EXPLAINED BY
          IDIOPATHIC_HYPERLUCENT_LUNG_RT_SIDE

*** REPORT 2 ***
------ ------ 0 — 0 INCR_DENS/BULGE/PROM/SUSPICIOUS_AREA_R_HT_BORD_
                      OR_PERICARD/PARASPINAL
------ EFFECT 5 — 6 INCR_LUCENCY_BOTH_LUNGS
CAUSE  ------ 5 — 5 CHRONIC_OBSTRUCTIVE_DISEASE_BOTH_LUNGS
CAUSE  ------ 0 — 0 IDIOPATHIC_HYPERLUCENT_LUNG_RT_SIDE
------ ------ 0 — 0 NODULAR_DENSITY_RT_LUNG_ADJACENT_TO_SCAPULAR_ANGLE

INCR_LUCENCY_BOTH_LUNGS
     EXPLAINED BY
          CHRONIC_OBSTRUCTIVE_DISEASE_BOTH_LUNGS
          OR IDIOPATHIC_HYPERLUCENT_LUNG_RT_SIDE
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Expert Subject E4

*** 2 SEC ***
------ ------ 5 -- 0 MALE
CAUSE  ------ 9 -- 0 OLD_AGE
------ EFFECT 5 -- 0 INCR_EXPANSION_BOTH_LUNGS
CAUSE  EFFECT 6 -- 5 EMPHYSEMA_BOTH_LUNGS
------ EFFECT 0 -- 0 ARTERIOSCLEROTIC_HEART
CAUSE  ------ 0 -- 0 NORMAL_IF_AGE_ADJUSTED
CAUSE  ------ 0 -- 0 SMOKING_HISTORY

INCR_EXPANSION_BOTH_LUNGS
     EXPLAINED_BY
          EMPHYSEMA_BOTH_LUNGS

ARTERIOSCLEROTIC_HEART
     EXPLAINED_BY
          NORMAL_IF_AGE_ADJUSTED

EMPHYSEMA_BOTH_LUNGS
     EXPLAINED_BY
          OLD_AGE
          AND_SMOKING_HISTORY

*** PROMPT ***

*** REPORT 1 ***
------ ------ 5 -- 0 INCR_EXPANSION_BOTH_LUNGS
------ ------ 5 -- 5 INCR_EXPANSION_R_LUNG
CAUSE  ------ 0 -- 0 BULLOUS_EMPHYSEMA_RT_LUNG
------ ------ 0 -- 0 BULLOUS_EMPHYSEMA_RT_UPPER_LOBE
------ EFFECT 0 -- 0 COMPRESSION_OR_CROWDED_LUNG_STRUCTURES_RT
------ EFFECT 0 -- 0 QUESTIONABLE_CARDIOVASCULAR_STRUCTURES/OUTLINE
CAUSE  ------ 0 -- 0 NORMAL_IF_AGE_ADJUSTED
------ EFFECT 3 -- 0 R_HILAR_TRIANGULAR_DENS
CAUSE  ------ 0 -- 0 R_LUNG_ORIGIN
CAUSE  ------ 0 -- 0 PARTIAL_OR_SEGMENTAL_ATELECTASIS_R_LUNG
CAUSE  ------ 0 -- 0 TUMOR_MASS_ABOVE_R_HILUM

COMPRESSION_OR_CROWDED_LUNG_STRUCTURES_RT
     EXPLAINED_BY
          BULLOUS_EMPHYSEMA_RT_LUNG

QUESTIONABLE_CARDIOVASCULAR_STRUCTURES/OUTLINE
     EXPLAINED_BY
          NORMAL_IF_AGE_ADJUSTED

R_HILAR_TRIANGULAR_DENS
     EXPLAINED_BY
          R_LUNG_ORIGIN
          OR_PARTIAL_OR_SEGMENTAL_ATELECTASIS_R_LUNG
          OR_TUMOR_MASS_ABOVE_R_HILUM
```

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*** REPORT 2 ***
------ ------ 6 — 5 EMPHYSEMA_BOTH_LUNGS
CAUSE  ------ 5 — 5 R_LUNG_EMPHYSEMA
------ ------ 0 — 0 BULLOUS_EMPHYSEMA_RT_LUNG
------ ------ 0 — 0 BULLOUS_EMPHYSEMA_RT_UPPER_LOBE
------ EFFECT 3 — 0 R_HILAR_TRIANGULAR_DENS
CAUSE  ------ 0 — 0 R_HILAR_NEOPLASM
------ EFFECT 0 — 0 QUESTIONABLE_CARDIOVASCULAR_STRUCTURES/OUTLINE
CAUSE  ------ 0 — 0 NORMAL_IF_AGE_ADJUSTED
------ EFFECT 0 — 0 QUESTIONABLE_MEDIASTINUM
------ ------ 0 — 0 R_AZYGOS_LOBE

R_HILAR_TRIANGULAR_DENS
     EXPLAINED BY
          R_LUNG_EMPHYSEMA
          OR R_HILAR_NEOPLASM

QUESTIONABLE_CARDIOVASCULAR_STRUCTURES/OUTLINE
AND QUESTIONABLE_MEDIASTINUM
     EXPLAINED BY
          NORMAL_IF_AGE_ADJUSTED
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Expert Subject E5

*** 2 SEC ***
------ EFFECT 0 — 0 MASS_R_LUNG_HT_BORDER
CAUSE  ------ 0 — 0 R_LUNG_MASS
CAUSE  ------ 3 — 0 R_MEDIASTINAL_MASS
------ ------ 5 — 0 MALE
------ ------ 5 — 5 INCR_EXPANSION_R_LUNG

MASS_R_LUNG_HT_BORDER
     EXPLAINED_BY
          R_LUNG_MASS
          OR R_MEDIASTINAL_MASS

*** PROMPT ***
------ EFFECT 5 — 5 INCR_EXPANSION_R_LUNG
------ EFFECT 0 — 0 SMALL_HEART
------ ------ 0 — 0 MASS_R_LUNG_HT_BORDER
CAUSE  ------ 0 — 0 CHRONIC_LUNG_DISEASE
------ EFFECT 0 — 0 LEFT_BREAST_LARGER_THAN_RIGHT
CAUSE  ------ 5 — 0 MALE
CAUSE  ------ 0 — 0 OLD_FEMALE
CAUSE  ------ 9 — 0 ROTATION

INCR_EXPANSION_R_LUNG
AND SMALL_HEART
     EXPLAINED BY
          CHRONIC_LUNG_DISEASE

LEFT_BREAST_LARGER_THAN_RIGHT
     EXPLAINED BY
          MALE
          OR OLD_FEMALE
          OR ROTATION

*** REPORT 1 ***
------ EFFECT 5 — 5 INCR_LUCENCY_R_LUNG
------ EFFECT 0 — 0 DECREASED_VASC_R_LOWER_LOBE
------ EFFECT 0 — 0 DECREASED_VASC_R_MIDDLE_LOBE
------ EFFECT 0 — 0 DECR_VASC_R_UPPER_LUNG
------ EFFECT 3 — 0 MASS_R_LOWER_LUNG_MEDIALLY
------ EFFECT 5 — 4 POORLY_SEEN_RT_HEART_BORDER
CAUSE  ------ 0 — 0 R_MIDDLE_LOBE_MASS
CAUSE  ------ 0 — 0 R_LOWER_LOBE_MASS
------ ------ 3 — 0 POORLY_SEEN_R_HILUM
------ ------ 0 — 0 UNUSUAL_MEDIAL_LOCATION_R_MAIN_DESC_PULMONARY_
                      ARTERY
------ EFFECT 0 — 0 INCR_DENS_R_UPPER_LUNG
CAUSE  ------ 0 — 0 R_AZYGOS_LOBE
------ ------ 0 — 0 EMPHYSEMATOUS_BULLAE_RT_UPPER_CHEST
------ EFFECT 6 — 0 FLAT_R_DIAPHRAGM
CAUSE  ------ 5 — 5 INCR_EXPANSION_R_LUNG
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CAUSE  ------ 0 — 0 CHRONIC_OBSTRUCTIVE_PULM_DISEASE
CAUSE  ------ 0 — 0 HYPOPLASTIC_R_LUNG
CAUSE  ------ 3 — 2 R_LOWER_LOBE_ATELECTASIS

INCR_LUCENCY_R_LUNG
AND DECREASED_VASC_R_LOWER_LOBE
AND DECREASED_VASC_R_MIDDLE_LOBE
AND DECR_VASC_R_UPPER_LUNG
     EXPLAINED BY
          CHRONIC_OBSTRUCTIVE_PULM_DISEASE
          OR HYPOPLASTIC_R_LUNG
          OR R_LOWER_LOBE_ATELECTASIS

POORLY_SEEN_RT_HEART_BORDER
     EXPLAINED BY
          R_MIDDLE_LOBE_MASS

INCR_DENS_R_UPPER_LUNG
     EXPLAINED BY
          R_AZYGOS_LOBE

FLAT_R_DIAPHRAGM
     EXPLAINED BY
          INCR_EXPANSION_R_LUNG

MASS_R_LOWER_LUNG_MEDIALLY
     EXPLAINED BY
          R_LOWER_LOBE_MASS
          OR R_MIDDLE_LOBE_MASS

*** REPORT 2 ***
------ EFFECT 5 — 5 INCR_LUCENCY_R_LUNG
------ ------ 5 — 6 DECR_VASC_R_LUNG
------ ------ 0 — 0 SMALL_RT_HILUM/HILAR_VESSELS
------ ------ 0 — 0 UNUSUAL_MEDIAL_LOCATION_R_MAIN_DESC_PULMONARY_
                      ARTERY
------ ------ 0 — 0 EMPHYSEMATOUS_BULLAE_RT_UPPER_CHEST
CAUSE  EFFECT 3 — 0 MASS_R_LOWER_LUNG_MEDIALLY
CAUSE  ------ 0 — 0 R_LOWER_LOBE_MASS
CAUSE  ------ 0 — 0 R_MIDDLE_LOBE_MASS
CAUSE  ------ 3 — 3 R_PARASPINAL_MASS
------ EFFECT 5 — 4 POORLY_SEEN_RT_HEART_BORDER
------ EFFECT 6 — 0 FLAT_R_DIAPHRAGM
CAUSE  EFFECT 5 — 5 INCR_EXPANSION_R_LUNG
CAUSE  ------ 0 — 5 CHRONIC_OBSTRUCTIVE_DISEASE_R_LUNG
CAUSE  ------ 0 — 0 COMPENSITORY_HYPERTROPHY_RT_LOBE
------ ------ 0 — 0 INCREASED_RIB_INTERSPACES_RT_CHEST
CAUSE  ------ 0 — 0 HYPOPLASTIC_R_LUNG

MASS_R_LOWER_LUNG_MEDIALLY
     EXPLAINED BY
          R_LOWER_LOBE_MASS
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          OR R_MIDDLE_LOBE_MASS
          OR R_PARASPINAL_MASS

POORLY_SEEN_RT_HEART_BORDER
     EXPLAINED BY
          MASS_R_LOWER_LUNG_MEDIALLY

FLAT_R_DIAPHRAGM
     EXPLAINED BY
          INCR_EXPANSION_R_LUNG

INCR_LUCENCY_R_LUNG
AND INCR_EXPANSION_R_LUNG
     EXPLAINED BY
          CHRONIC_OBSTRUCTIVE_DISEASE_R_LUNG
          OR COMPENSITORY_HYPERTROPHY_RT_LOBE
          OR HYPOPLASTIC_R_LUNG
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